Industrial Medicine

for

Physicians and Surgeons in Industry

Volume 5

The Pneumonokonioses (Silicosis)

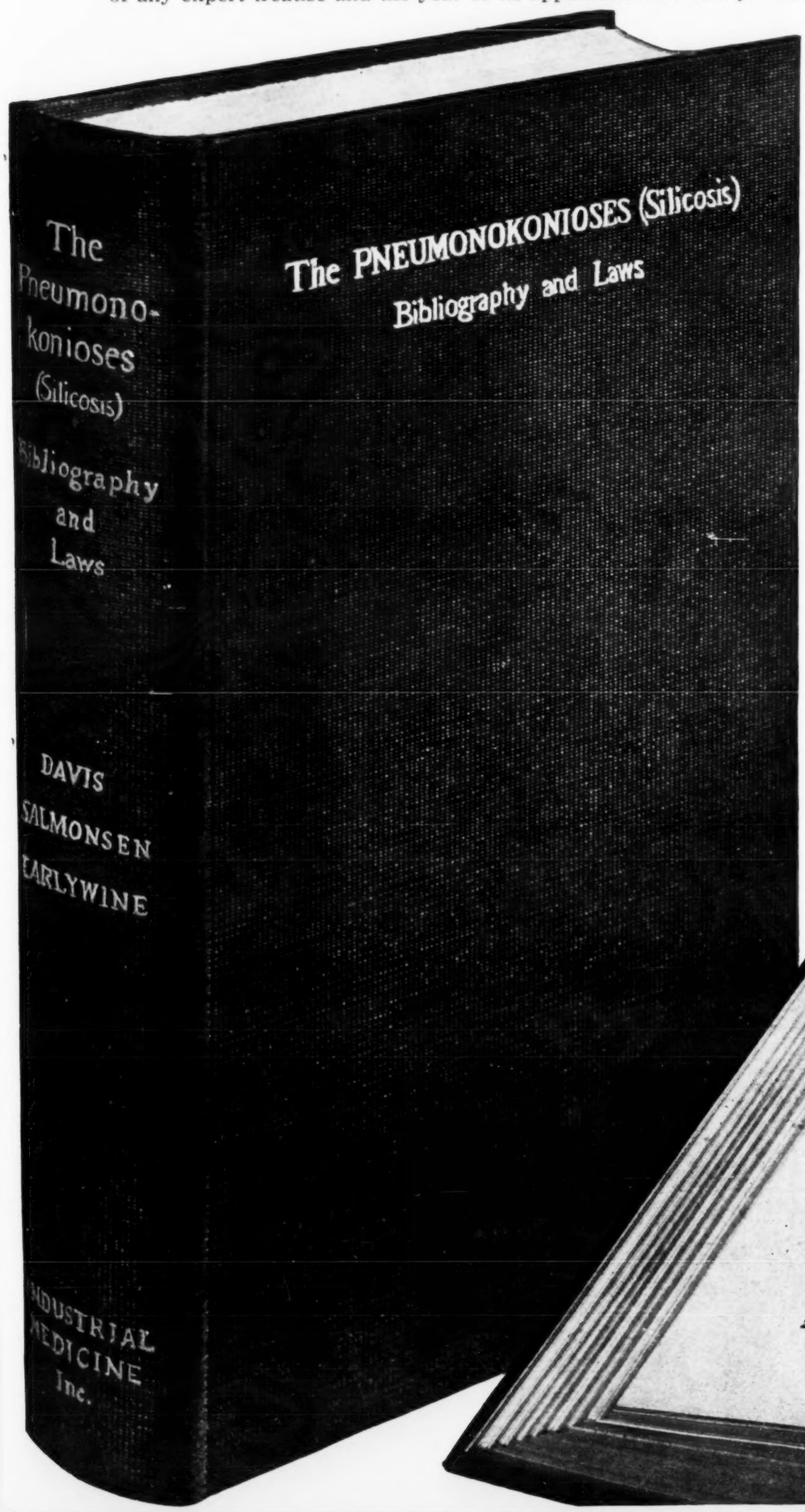
Bibliography *and* Laws Book No. I

All the References of the World's Literature
--- from 1556 to December 31, 1933 ---

Review from **"Staub"**, (Dust), 1, No. 1:167-168, April, 1936, done by Dr. Ing. O. M. Faber, Berlin.

No. 115. Bibliography and Laws upon Silicosis. **(The Pneumonoconioses (Silicosis) Bibliography and Laws),** by Geo. G. Davis, E. M. Salmonsen and J. L. Earlywine, published by **Industrial Medicine,** Chicago, Ill., 1934, 482 pp. ($7.50).

The **Forword** is by E. R. Le Count, Chief of the Department of Pathology, The University of Chicago. The book has two main divisions. Part I is a Bibliography of the collected literature of the world upon the **Pneumonoconioses (Silicosis).** Beginning with the **De Re Metallica** of Agricola (1556), it ends with 1933, inclusive of 2,768 sources, whose title, author and exact citation are given. The works are chronologically arranged. Through subdivision into different groups, the works of authors appearing in the same year are clearly and immediately perceived. Within the groups the works are still arranged in alphabetical sequence by authors. These 276 pages of comprehensive bibliography are followed by a complete **author—**and **subject—index.** Besides, the location (orientation) of any expert treatise and the year of its appearance are easily made out at a glance.



In Part II, the Laws of the several States of the Union in North America, as relating to Occupational Diseases, the injuries they cause, and any pertinent material, are all represented. The plan and sub-divisions by which the situation can be ascertained for any one of the 48 states of the United States of America, are readily apprehended. In several cases corresponding paragraphs have cross-references. Especially valuable and complete is this legal survey for ascertaining any characteristic verdict (judgment) in respect of a pneumonoconiosis, showing the basis of the action, the conditions giving rise to the action, as well as the principle involved in the decision—for any state in the Union. The book closes with an alphabetical summary of the enterprises concerned in the juridical distinctions (or decisions.)

Examination of the book compels recognition of the extraordinary care and pains taken by the compilers. Random tests here and there show that for a space of 377 years this bibliography has attained a completeness not hitherto arrived at. Probably no branch of any other science has at its disposal so flawless and richly extensive a bibliography. It is authoritative not only for physicians and industrial experts, but also for physicists, engineers, and employers of industry. The format is entirely appropriate; for the arduous work involved the compilers should receive the fullest recognition.

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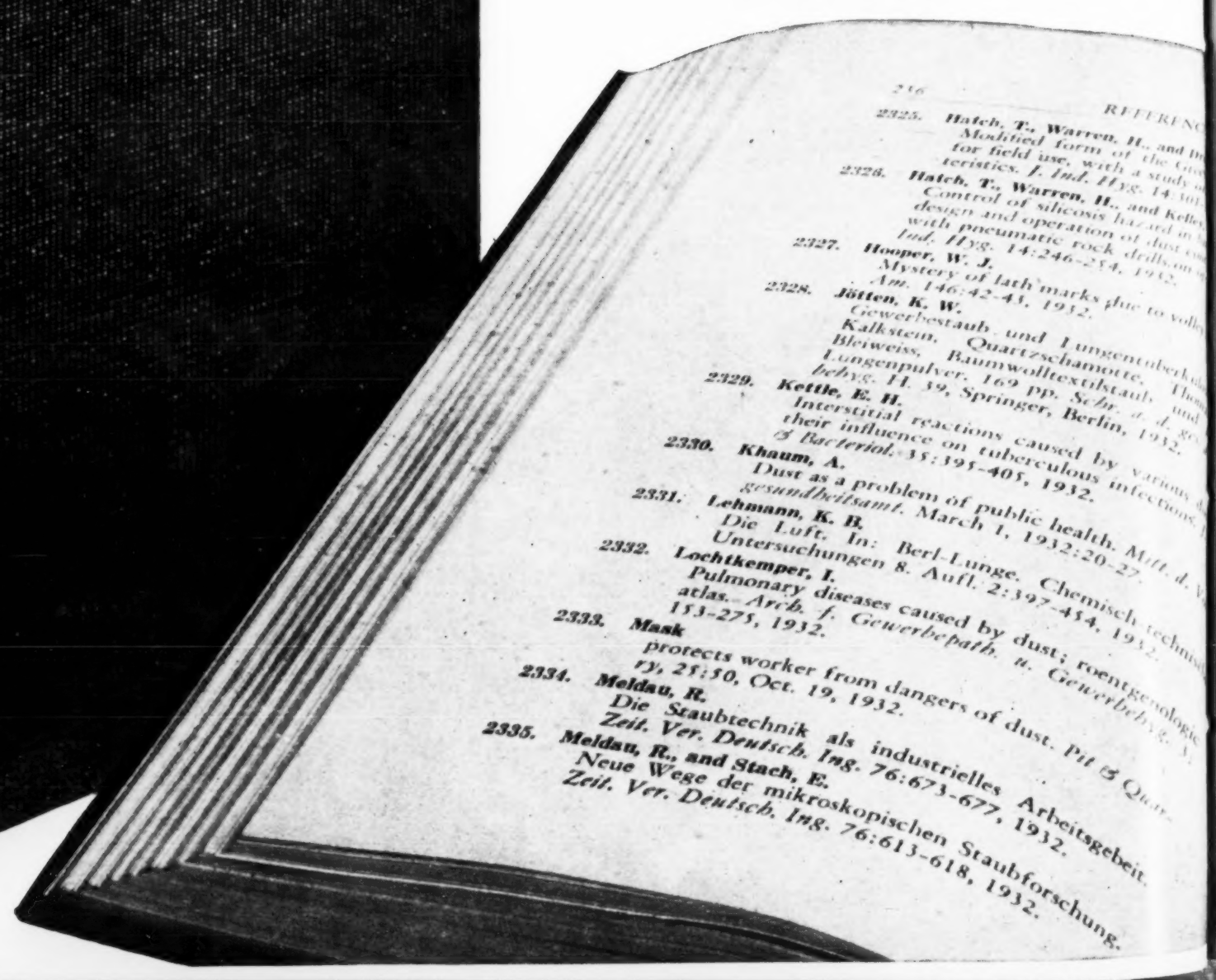
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The Law, the Science and the Economics of Industrial Health

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The Injection Treatment of Hernia

—The Current Objections—

by

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New York City

IN THE course of almost daily demonstrations of the injection treatment of hernia to members of the medical profession, I am constantly being asked certain definite questions as to possible complications and difficulties. These same questions recur with such regularity as to appear like collusion among the inquirers, or to suggest some widespread propaganda against the method to which they have all been exposed.

The fact, however, that these questions or objections are "natural" (arising logically from considerations of the anatomy and processes involved) suggests instead that they are simply the obvious questions and doubts of the medically trained mind. The truth probably is that these doubts are at first spontaneous, but are then reinforced by discussion with other doubters. Here and there, too, a bit of self-interest may be thrown in, as when a surgeon permits personal concern for surgical fees to intensify his mere doubts into firm and vigorous objection. At this point, a self-interested and unethical propaganda does enter, and is today undoubtedly a real factor in discouraging a general and impartial consideration of this method of treating hernia.

My own inquirers have, however, quite invariably displayed a genuine desire to know, and while often conservative and critical in attitude, have clearly come to my office to learn what actual experience with the treatment indicates. Favorable clinical experience is of course the proper and final answer to questions and doubts, and fortunately it does not confirm the fearful expectations so widely current. The injection treatment of hernia does have its difficulties and complications, but strangely enough they are seldom those so generally anticipated, and fortunately too, they are neither frequent nor serious in character.

I am able to speak with peculiar authority in this matter, for in addition to my own seven years of direct experience in more than 800 cases, I have carefully reviewed the entire literature and have recently corresponded with nearly 200 American workers with the method, and find that my own experience is also their general experience in some 12,000 cases.

Yet such is the suggestive effect of being exposed to continual doubting and questioning, that one keeps looking for the very sequelae which everyone else looks for, this despite the definite lesson of wide experience that the foreseen complications do not occur in fact. As a result, I have been especially watchful for just such untoward difficulties and results, but still have not found them in actuality.

Those doctors who come to me for demonstrations of technic and results, have usually read my previous articles, 10, 11 or have otherwise gained an approximately correct idea of the method employed. They, therefore, do not come with objections arising out of serious misconception of the method. Patients, however, often bring in reports of warnings from other doctors that we are injecting paraffin, "glue" or even "poison," that the results may be very serious, and of course that the method is wholly worthless. A rectifying word as to the general method and theory of the treatment therefore seems desirable before presenting specific replies to the several better-considered questions and doubts.

ONLY reducible hernias are properly treated by the injection method, and these are kept continuously reduced during treatment by a suitably fitting controlling truss, worn throughout the period of treatment and for a very few weeks thereafter while the repair is gaining firmness and strength. Strangulated, incarcerated and other irreducible or uncontrollable cases are not to be treated by the injection method.

Modern injectants are mildly irritant solutions designed to stimulate seroplastic proliferation of fibrous connective tissues which by adhesion and obstruction effect closure of hernial openings and the reinforcement of weakened and yielding muscular and fascial walls and integuments. After considerable experiment, I use in my own practice a commercially available hydro-alcoholic solution of plant gallic and tannic acids (Galtanol-Loeser). A preliminary local anaesthetic is first injected, usually procaine or some equivalent, without adrenalin.

The patient is placed prone on the table, with a slight Trendelenburg tilt, and the hernia *completely* reduced. Careful observation during reduction will often help in locating the precise positions of hernial rings. The area to be injected is shaved and swabbed with antiseptic as for a surgical operation. In indirect inguinal cases, the first injections are given into the deeper and medial margins of the internal ring, with subsequent injections along the canal towards the external ring which is closed last of all. About 15 injections given two or three a week are required for the *thorough* repair of an ordinary inguinal hernia.

For inguinal cases, a 22 gauge, 1½ inch hypodermic needle is best (preferably with a bead guard near its shank). After inserting the needle, always aspirate, and if no blood appears, inject the procaine. If blood appears in the syringe from aspirating, draw the needle back and shift its point in the tissues. The needle is left in situ for the few minutes until the anaesthetic takes full effect. Then the syringe containing the prescribed dose (2 cc. for Galtanol) is attached and the injection given *very slowly*. The needle is then withdrawn, but the patient is kept prone for perhaps another 10 minutes before replacing the truss and permitting him to rise and proceed about his normal activities.

Questions and Objections

AMONG objections to the injection treatment of hernia, the general feeling that it is "blind" probably comes first. How can one know the precise condition of the hernial lesion and must one not simply "guess" at the proper placing of the injectant? This doubt is particularly forceful to the surgeon, for in the operative treatment he does fully disclose the internal condition, and needs must, to permit the precision of work which surgery demands. He simply must see what his dangerously keen scalpel and needles are doing. So he naturally thinks in terms of precision, overlooking the fact that the things about which he must be so precise are not involved in the injection treatment at all. The hypodermic needle wounds the tissues so insignificantly and at such wide intervals that its own direct effects may be quite ignored. The repair is also effected by a localized, yet sufficiently general, tissue growth which demands no precision in placing at all comparable to surgical requirements.

On the other hand, as will presently be shown, the injection treatment is not nearly so "blind" as would superficially appear. The anatomic relations in inguinal and femoral hernias are quite definite, and can ordinarily be palpated externally or (in inguinal cases) explored by invaginating the scrotum. Also, as one proceeds with treatment, indurations develop along the hernial canal, become palpable and so guide the course of injections. This guidance is particularly useful in treating incisional hernias, as the normal relations of the structures have been altered and one must often guess as to points of weakening. One therefore proceeds tentatively along the cicatrice, noting results and continuing injections at the weak points which remain or may subsequently develop.

There is also real guidance in the "feel" of the needle as it passes through the several tissues. Knowing the anatomy, and with a little experience, one can literally "feel one's way" and know just where the needle point is. The denser muscular, fascial and connective tissues also resist the injection of solution quite appreciably, and one should always inject into such resistant tissues, never into cavities.

2. A related question of my professional inquirers is: "How can one locate the internal inguinal ring?" It is true that unless it is very large and the patient spare, one can scarcely palpate it. It is also not always possible to locate it by watching during reduction, nor can it normally be reached with the finger invaginating the scrotum and exploring along the canal through the external ring. Then it is necessary to resort to topographic means: the internal ring being about three-eighths of an inch above the mid-point of a straight line drawn from the pubic spine to the anterior superior spine of the ilium (see Fig. 1). Once located, it can be outlined on the skin for future guidance by means of mercurochrome or some other lasting stain. Similarly, mercurochrome spotting of each point of injection as made, may serve as a helpful guide in later injecting, besides being a good antiseptic precaution. Locating the external ring is usually no problem, as it is ordinarily apparent by both palpation and scrotal invagination.

3. Coupled with another question of location, "How deep should one inject?" is that anticipation of trouble, "How can one avoid injecting into the peritoneal cavity and viscera?" and further, "Will not peritonitis set in and visceral perforations and adhesions result, with most serious consequences, even danger to life?"[12]

These questions have already been answered practically in setting forth the rule, "Inject only into firm tissues, resistant both to the needle and to the flow of the injectant." Thus cavities are definitely avoided, including the peritoneal cavity and its yielding visceral contents. Furthermore, with its layer of subcutaneous fat and several deeper layers of muscle and fascia, the abdominal wall of the inguinal region is thicker than is realized by those inexperienced in surgery or injections. In fact, the 1½ inch needle is ordinarily inserted (obliquely) nearly to its shank when injecting the deeper muscular wall of the inguinal canal (see Fig. 2).

The beginner with the injection treatment often fails to appreciate this, and permits his fear of penetrating into the abdominal cavity to lead him into giving too superficial treatment. The resulting closure is then incomplete, and the viscera may slip out behind the repair. This introduces a real difficulty, for the dense and tough superficial induration is hard to penetrate with the needle and one finds it's hard to judge from the feel whether the needle has entered a cavity or merely less resistant tissue behind this tough layer. Fig. 2 will suggest the proper depths of injection in normal cases, but consideration must always be given to abnormal obeseness or spareness in judging depths. Judgment and the "feel" must also be relied upon largely in incisional cases (including surgical recurrences) as the normal anatomy has been disturbed and the abdominal wall may prove unexpectedly thin. In short, knowledge of anatomy, thorough training in technique, and experience are all important in this as in other treatment.

Emphasizing the rarity of death following the injection treatment, only two fatal cases are

known. Bratrud has recently reported a case (treated by someone else) in which 16 minims of Thuja were injected, the ileum being perforated, with peritonitis and death resulting.[15] The other fatal case (reported by Goldhahn) was treated by an unlicensed practitioner.[5] He reports two cases suffering from fecal fistula following such irregular treatment by injection, and a third dying from sepsis and a heart condition. The histories in these cases were quite inadequate, but the symptoms in all three cases suggest strangulation, sepsis and intestinal perforation, with perhaps forcible reduction and subsequent treatment by injection. No responsible and informed physician would today treat such cases by injection, for strangulation is always given as a decisive contra-indication of the method. It should also be noted that these unlicensed German "specialists in hernia" employ an inadequate solution, merely alcohol, the method being in fact known as that of "alkoholinjektionen." The resulting tissue proliferation is both slow and slight, necessitating a large number of injections over a period of a year or more. With our modernized and highly efficient solutions, comparatively few injections are needed, with the time cut to only a few weeks.

Wolfe[6] reports that in experimental work with rats, injections were accidentally made into the peritoneal cavity, the resulting inflammation being rapidly fatal, while autopsy revealed many firm adhesions. But his injection doses, considering relative body weights, were from 20 to 30 times those employed in treating human beings. Furthermore, experience proves that, in practice, there is no difficulty in avoiding injection into the human peritoneal cavity. The fear of peritonitis and intestinal adhesions is hardly warranted by the facts of experience, the incidence being altogether negligible. And with only two fatal cases reported in some 30,000 recorded cases, the danger of death may also be considered quite literally non-existent. This is even better than the very fine record established by recent hernial surgery, approximating a third or a half of one percent in non-strangulated cases. The surgical death rate in strangulated cases is of course much higher.

In two recent cases reported to me, but as yet unpublished, strangulation has occurred during the course of the injection treatment, with that treatment partly responsible for the condition. In each case the internal ring has been reduced in size by the proliferative growth induced by the treatment. In each case also, the patient admitted not keeping his truss on. The uncontrolled hernia prolapsed through the smaller and tighter ring, and strangulation ensued. There is a real danger here, to be overcome by emphasizing to the patient the importance of his cooperation in wearing his controlling truss. Fortunately this complication is seldom met with, and, being due to carelessness, should be readily avoidable.

4. Presenting another aspect of this problem of avoiding injection into the peritoneal cavity, are those frequent questions, "Does one inject into the hernial sac?" and "How can one prevent solution injected into the sac from draining into the peritoneal cavity and causing dangerous visceral adhesions?"

The modern technique does not aim to inject into the hernial sac. In fact, as Gray pointed out[7], the sac of a fully reduced hernia is completely collapsed, and to find and inject it with a needle again and again would be quite beyond human skill. Usually, too, the sac in indirect inguinal cases is involved with the cord, and is therefore avoided through the deliberate avoidance of the cord. A century ago, Velpeau[1] did attempt this very technique of injecting the sac, but he had to make an open incision to find the sac, which he then flushed with an iodine solution, which he withdrew as well as injected through a cannula, while an assistant applied pressure at the neck of the sac to prevent draining of the irritant solution into the abdominal cavity. Furthermore, Velpeau only "injected" thus once in treating a hernia.

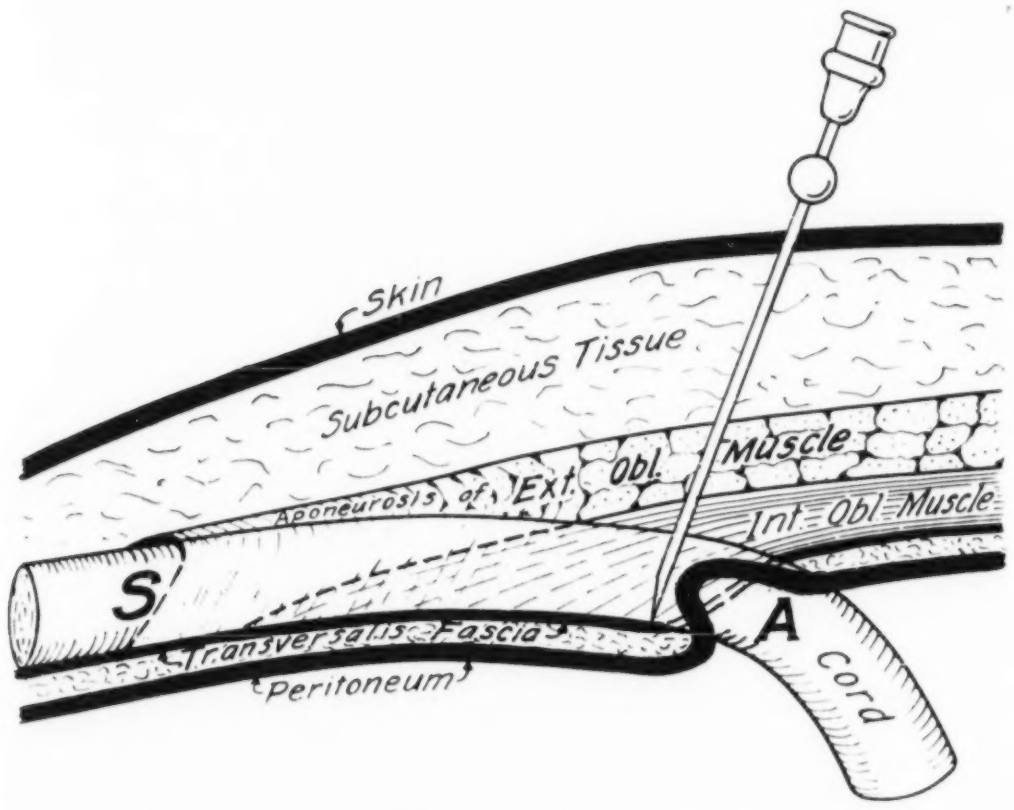

Fig. 1. The Ordinary Abdominal Hernias
A—Internal Inguinal Ring showing slight hernial sac
S—External Inguinal Ring

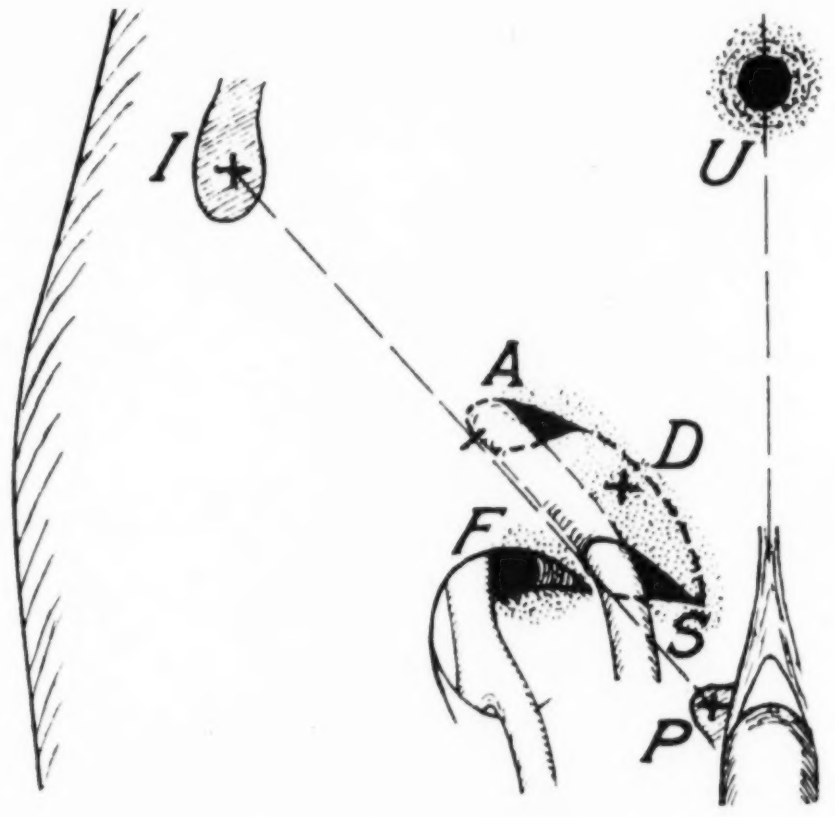

Fig. 2. Diagrammatic Section along Inguinal Canal
Hernial rings—black;
A—Internal Inguinal Ring;
S—External Inguinal Ring;
D—Direct Inguinal Site;
F—Femoral Ring;
U—Umbilical Ring
Injection areas—Dotted

Later technicians, such as Heaton[2] and Warren[3], carefully injected the canal *outside the sac*, again employing a single injection (except in stubborn cases), but that injection exceedingly drastic. The modern technic uses an ordinary hypodermic needle to give an extended series of injections, employing mild irritants, and these introduced into resistant muscular and fascial tissues instead of into cavities such as the sac and canal. Solution so injected diffuses but slowly, and its seroplastic effect is quite local.

Yet doubtless the sac is accidentally injected on rare occasions, perhaps because of incomplete reduction of the hernia, or the presence of fluid within the sac preventing its complete collapse, or perhaps the needle has passed through the sac to reach deeper tissues. I have certainly administered in excess of 12,000 hernial injections, and in only three or four instances have I suspected that the solution has drained from the sac into the peritoneal cavity. For the patients experienced immediate crampish pains in the region, lasting from a quarter to half an hour, but with no further observed harm of a permanent character. In no case has this occurred twice with a given patient. If the immediate reaction was really due to drainage into the peritoneal cavity from the sac, the probable effect was to cause early adhesions and obliteration of the sac by closure, thus helping prevent recurrence of such drainage. In any event, no harm was done beyond some moments of pain, and this with negligible rarity.

5. A somewhat related question is asked less often, "Is there not danger from the injection or absorption into the blood stream of an irritant, perhaps sclerosing, solution?"

It is the accepted practice to aspirate after placing the needle and before injecting, and if blood appears, to withdraw the needle somewhat and shift its point in the tissues. In the inguinal region, the only important vessels are the inferior epigastric artery and veins, but they are deep and give little trouble. The femoral artery is well removed from the site of femoral injection, and the femoral vein, though just lateral to the femoral canal, is palpable and readily avoided. Not so the obturator artery, however, for in perhaps a third of cases, its anomalous course over the femoral fossa demands careful avoidance. But the injection of these and minor blood vessels is always readily avoided by aspirating.

This technic has another advantage, it avoids the reinjecting of an area while active proliferation is in progress. For in the early process of seroplastic repair, the granulation tissues are suffused with blood which appears upon aspirating. Later in the process, the hard indurarion may be palpated and offers strong resistance to both needle and injectant, thereby guiding avoidance of the actively growing tissues. This hard induration presently softens as the fibrous connective tissue completes the repair.

There must, of course, be some obsorption of solution from injected tissues into the blood stream. However, such absorption is slow and slight, especially if the injection is definitely into muscular and fascial tissues and administered very slowly, with a lapse of 10 or 15 minutes before applying truss pressure. With proper hernial injectants, such absorption is merely wasteful, not harmful, for obviously a proper hernial solution is nontoxic, non-allergic and non-sclerosing. The use of solutions designed for treating varicose veins should therefore be strictly barred in treating hernias, for their effect is specifically and purposely sclerosing. While there are several formulas and commercially available solutions for hernial injection, only those should be used which have been thoroughly tested for absence of systemic effects.

6. There is undoubtedly a general feeling that the injection treatment somehow involves a lack of control over the growth of repair tissue, giving too little here, too much there, that the hernial injectant diffuses through the tissues so that the repair is frequently misplaced, etc.[12] Strangely enough, this objection usually comes from surgeons, who certainly have no control over the growth of repair tissue once their incisions have been closed.[14]

The objection might well have been pressed against the technics of the early workers, Velpeau, Pancoast[4], Heaton and Warren, with their single drastic injections into the hernial cavities. They certainly had no control over the resulting tissue repair, except that if it proved insufficient, they might give further injections. With the modern technic of a series of hypodermic injections, extending over several weeks, repair is always closely under control. Proliferation is quite definitely localized by injection into muscular and fascial tissues, never into cavities. If the solution be properly concentrated (fully effective with say 2 cc. dosage), there is little diffusion, especially if the technic is observed of not applying truss pressure until 10 or 15 minutes after injection. Within a few days the local induration indicates the extent of the seroplastic action, guiding further treatment. As repair proceeds, it can therefore be placed and added to as needed. With the injection treatment, progress can be watched and controlled without difficulty.

The danger lies rather in insufficient treatment than too much. For some subsequent tissue resorption must inevitably occur and should be allowed for by an initial overtreatment. Then by watching the case at intervals for some months, any resulting weakening and threat of recurrence can be anticipated by reinforcing injections. The injection treatment is thus in fact the one treatment with a really practical control over hernial repair, and if consistently followed up, over recurrence as well. It has even been used most successfully in supplementing surgical repairs which are weakening through resorption or initially incomplete closure. Its success in cases of surgical recurrence is now commonplace.

This of course raises the important question of the relative recurrence rates following the injection and surgical treatments. The frank and truthful answer is that as yet we have but little exact and dependable information as to the recurrence rate after the injection treatment. Being ambulatory, that treatment is usually given by private practitioners who have few facilities for following up cases. Hospitals and insurance carriers could make the needed study to much better advantage. For myself, I cannot even pretend to have kept track of enough of my cases for a sufficient length of time to warrant my even estimating the recurrence rate. I am quite sure that to date the only significant figures are those of Bratrud from the University of Minnesota Hospital Clinic[15]. He reports only 4% recurrences in some 406 hernias,

with only three failures to effect complete closure. Of course this high rate of success is not likely to be maintained by the general run of less skilled workers with the method, but on the other hand, recurrences usually yield so readily to reinforcing injections, that the rate of final success will probably approximate his figure.

7. Patients unfamiliar with hernial and related anatomy are not likely to ask such technical questions as those above discussed. But they are often agitated over another group of inferred dangers over which members of the profession also show frequent and more specific concern, namely, "How can one avoid injecting the spermatic cord?" and "Will not injection of the cord cause local inflammation, proliferation and closure, as well as a whole train of related sequelae, such as orchitis, epididymitis, hydrocele, atrophy of the testicles, impotence and sterility?"

It is, of course, undesirable to inject the cord, and it can ordinarily be readily avoided. Normally it lies closely alongside the inguinal (Poupart's) ligament which can be palpated without the slightest difficulty. Injections are simply made a half-inch or more medially from the ligament to allow for the thickness of the adjacent cord which is less obviously palpable. Occasionally, working too closely, one does encounter the cord and can even feel it yield by rolling aside as the needle presses at its tough integuments. And then, infrequently, one probably penetrates the cord, and even injects it. A local inflammatory reaction naturally follows, sometimes reported as extending towards the epididymis, but without either epididymitis or orchitis.

Wolfe[6] reported the results of repeated injections into the spermatic cord of a dog, an experiment designed to reveal just what dangers such injection might involve. He says, "Thirty days later the cord and testicle were removed and several serial sections made from the cord and testicle. Upon opening the inguinal canal the tissue was found firm and resistant. The infundibuliform fascia was closely adherent to the surrounding tissue and the contents of the fascial tube slipped out easily.

"The biopsy revealed the following: 'To gross examination the testicle, the vas deferens and other tissue show no definite lesion. Microscopic sections of the testes are normal. Several sections passing through the vas deferens show it perfectly preserved. Its lumen is patent, and the epithelial lining is intact. In the tissues surrounding the vas, but exterior to its wall, there is a mild subacute inflammatory reaction. There is considerable infiltration with leucocytes, principally mononuclear, but with an occasional polymorphonuclear and some proliferation of fibrous connective tissue. A vessel in the inflamed area is filled with a recent thrombus, which shows beginning organization . . . Injection into the spermatic cord need not necessarily cause obliteration of the vas."

Clinical experience is also very reassuring, for among some 12,000 cases of nearly 200 American workers with the method, none were reported as resulting in impotence or sterility, although the question was specifically raised with them all. Two reports ascribing such results to the injection treatment have, however, appeared in the last two years in the *Journal of the American Medical Association*. The first[9] referred to a man of 58 who had suffered spinal fracture and other injury, then "had an operation for double hernia following some injection treatment" and over a year and a half later became impotent and noted atrophy of the testicles. Both results are familiar following hernial surgery, and might well have arisen from the spinal fracture, yet the *Journal* editor (rather overzealously, one would say) ascribed the sexual disability solely to the injection treatment. The second report [13] gave no histories or other details, and private correspondence with the reporting physician revealed loose reliance on complaints from patients, without any confirming tests or examinations, nor the consideration of other possible causes, physical or psychological. The one confirming observation was that impotence was reported only in those who had been treated on both sides.

There is obviously the theoretical possibility that sexual disability or disturbance might ensue from pressure on nerves, blood vessels or the vas itself, by repair tissues induced by either surgery or injections. Truss pressure might effect the same result. Apparently the immediately pertinent problem is merely that of relative incidence following each type of hernial repair or control. The record of surgery in this respect is not serious, and that of the injection treatment to date seems ever more negligible. Naturally any careful and impartial study will consider other possible causes, and will particularly note prior conditions, such as undescended or atrophied or atrophying testicles, etc. I have myself come across a number of testicles already atrophied in hernia cases, and have made it a point to call the patient's attention to them, if only as a measure of professional protection. In cases of undescended testicles, I would not treat by injections, the condition obviously demanding surgical exploration and care. Needless to say, any case of atrophy, impotence or sterility traceable to the injection treatment should be most carefully studied and fully reported, particularly as to improvement in the technic of avoidance.

The Observed Complications

"IF THEN, these expected complications do not appear at all or with negligible rarity, what complications actually do appear? Surely the injection treatment of hernia does not violate all precedent by having no complications whatever?"

Two complications have been observed, an occasional hydrocele, and a more frequent inflammatory reaction local to the injected area. Two others have been reported, namely abscess and skin ulceration, but the former was definitely traceable to an outside source of infection, and the latter to truss chafing, appearing even before the injections began and disappearing with the removal of the truss. Necrosis and sloughing might be expected from injection of an irritant into fatty tissue, a very possible occurrence, but one not observed clinically. The solutions now in use are themselves definitely antiseptic, and with proper aseptic technic there should be no occasion for infection.

The hydroceles have been rare and transitory. In more than 800 cases of my own, I have had only three hydroceles, two of the scrotum and one of the cord. These hydroceles all subsided complete-

ly within two or three weeks, without any special treatment and without discontinuing the regular injections. Rice[8] also reports occasional hydroceles, subsiding in from one to six weeks. His careful studies indicate that these hydroceles result from the accidental injections of the hernial sac, as he found them in the area of, and extending only as far as, the observed hernial sac.

Occasionally an aseptic inflammation and soreness appear in the injected area. Then the fitting of the truss should first be inspected to make sure that undue pressure or chafing is not causing the trouble. The dosage should also be reduced and then tentatively increased as tolerance develops. The patient should also be advised, if soreness persists, to alleviate it with cold packs. Any such inflammation and soreness usually subside within a day or two and have never proved serious, except perhaps as a cause for alarm to the nervous patient.

Contra-Indications

IRREDUCIBILITY is always a definite contra-indication. Strangulation is definitely a surgical indication, as sepsis usually sets in quickly, and only surgery can explore and treat the condition with sufficient promptitude. Hemophylia and hyperthyroidism are also usually cited as contra-indications, but I have not had any such cases with hernia. On the other hand, I have found that diabetes and cardiac conditions are *not* contra-indications. Physicians are wisely cautious about injections in diabetic cases, but in those I have observed or had reported, the diabetic condition has tended to clear up, probably from the general improvement in health resulting from the relief of the hernia. I should perhaps qualify by stating that I have not tested in diabetic cases any but the solution I regularly use, and other solutions may not have similar effects. In cardiac cases, avoidance of organic shock is of course necessary, but observance of the rule—extreme slowness of injection—apparently fully obviates this danger.

Conclusions

THE injection treatment of hernia, with the technic and solutions now generally in use, is singularly free from complications, only rare and transitory hydroceles, and occasional (but controllable) inflammatory reactions being even noteworthy.

2. Modern hypodermic technic overcomes the various anticipated difficulties of the method, such as "blind" and uncontrolled treatment, penetration of or drainage into the peritoneal cavity, etc.

3. Animal studies and clinical experience indicate that the danger of sexual disturbance and disability is negligible. The danger of death from the injection treatment may be considered nil.

4. The contra-indications are irreducibility, strangulation, hemophylia and hyperthyroidism, but not diabetes nor cardiac condition.

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A.M.A. on Injection Method

IN MARCH, 1936, the Council on Pharmacy and Chemistry of the American Medical Association addressed a questionnaire to a selected list of hospitals, with a view to obtaining data on the injection treatment of hernia.* This method is now being exploited actively to the medical profession by many purveyors of mixtures of one type or another, used in this method of treatment. In the questionnaire, information was sought particularly as to the extent of this practice, its safety and its effectiveness, the extent of unfavorable complications, and the nature of such complications.

Replies, which were received from most of the institutions addressed, indicated that the method is not used in the majority of the hospitals consulted and that it is considered safe and effective by those using it, although many qualified their opinion as to safety and effectiveness by specifying careful selection of cases. Several unfavorable complications were recorded, including mild infections, the development of fibrous masses of cutaneous tissue, and failure of the method to correct the hernia. In addition, there were some instances of painful scarring, occasional cases of swelling of the testis, and a few instances of subsequent impotence. In two cases, gangrene followed the use of the method.

The solutions used by various observers included sodium morrhuate; a mixture of phenol, alcohol and oil of thuja; and several mixtures containing tannic acid or tannic acid derivatives. In some of the institutions, preparations of proprietary character, such as the Pina-Mestre solution, Galtanol and Proliferol, were used.

Opinions varied as to the number of cases suitable to this method of treatment. In those most favorable, from 12 to 15% of cases were treated by the injection method. Other institutions reported that the majority of cases of hernia in the outpatient clinic were treated by this method.

Several reports concerned surgical operations on patients who had been formerly treated by the injection method ineffectively. The surgeons re-

*Report of the Council on Pharmacy and Chemistry, PAUL NICHOLAS LEECH, Secretary, in *J. A. M. A.*, September 26, 1936.

port, however, that the previous injections had not made later surgery more difficult.

One of the complications involved in the introduction of the method has been the attitude of the insurance organizations, workmen's compensation boards and similar groups, in relationship to bearing the cost of such a procedure. Another complication has been the fact that the method has been taken up by osteopaths and even by laymen, and that some of the difficulties that have arisen are due to the results of such non-medical use of the injection method.

In some institutions in which physicians especially well trained have been put in charge of the injection method as administered in the outpatient clinic, the results seem to be quite satisfactory not only to the special worker but also to the members of the surgical staff who have followed these cases. The most unfavorable opinion seems to be that there may be more recurrences attending the injection of the indirect inguinal hernia than follow under adequate operative care.

It must be borne in mind, however, that these are ambulatory patients and that this type of care permits continued industrial employment as well as releasing a considerable number of beds for patients definitely requiring surgery.

Subsequent to the issuance of the questionnaire by the Council on Pharmacy and Chemistry, a letter was addressed to *The Journal of the American Medical Association* by some 50 physicians in good standing who have been using the injection method, citing their opposition to some of the criticisms contained in the questionnaire and emphasizing the fact that suitable selection of cases, injection by experienced practitioners, and suitable control of the patients treated make the injection method just as safe as, if not safer than a surgical procedure. These 50 physicians are convinced that the injection method is applicable and effective in the majority of cases of hernia, that it is often applicable when surgery is inadvisable and vice versa, and that it is just as free from complications and dangers as are surgical procedures. It is their opinion that the method will develop greater usefulness as it is more definitely understood, as the solutions used are more capably investigated and standardized, and as the limitations and uses of the method are more completely worked out.

With this point of view the Council on Pharmacy and Chemistry is inclined to agree. Nevertheless, the following considerations should be borne in mind not only by those expert in the injection method but also by the medical profession in general: The attempted cure of hernia by the application of the method of adhesive inflammation is not new. The method has, however, failed to establish itself as a routine method for the treatment of hernia and is still in an early experimental stage. In view of these facts, surgeons who practice this method should realize the dangers from an ethical, a legal and a financial point of view.

Those who inject into the bodies of their patients mixtures of unknown composition, not suitably standardized by any unprejudiced agency, and capable of bringing about definite pathologic changes in living, healthful tissue, may be subjecting themselves to unwarranted risk of legal difficulties should unfortunate results ensue. Those who continue to experiment with the injection method for the treatment of hernia with a view to standardizing this method will do well to use only such mixtures as may be prepared under their own direction in the laboratory of the hospital with which they may be associated, or mixtures prepared by competent pharmacists according to prescriptions prepared by the physician himself.

Occupational Diseases

The New Program Under the Ohio Department of Health

By EMERY R. HAYHURST, M.D.
Chief of the Division of Hygiene, and Consultant in Occupational Diseases

AS A BACKGROUND, it should be said that a Division of Industrial Hygiene was created in the Ohio State Board of Health as far back as May, 1913, when an extensive survey of industrial health hazards and occupational diseases occurring in the state was made, covering a two-year period.* This was at the instigation of a joint resolution of both houses of the legislature in 1913, and a commensurate report was made to the legislature at its next session in 1915. Following that time, the Division was continued with the personnel of one physician, a consulting physician, and a stenographer-clerk up to 1932, when its work was contracted to a Bureau of Occupational Diseases with only the author, acting as consultant, and the assistance of a part-time stenographer-clerk.

It has been my good fortune to have been associated with this field of work ever since its beginning in Ohio, first as Chief of the State Survey and later as Consultant upon various problems arising. During these 21 years, since the State Survey was made, the limited personnel and facilities nevertheless maintained a check on all occupational diseases reported, and pursued quite a number of investigations and researches which were published from time to time.

I should point out that the Legislature of 1913 passed what is known as the "Occupational Disease Reporting Law" which requires that every physician shall report to the State Director of Health, upon a specified form, any case which he believes to be occupational in nature. Ohio has always enforced this law, with the result that for a good many years more official reports of occupational diseases have been made in this state than in any other state, and in some years more than in all other states combined. Even so, the total number of occupational diseases reported for the 23-year period has amounted to only 19,343. Until 1921, when compensation for certain listed occupational diseases began, the average number of cases reported each year was 322, but since that date the yearly average has been 1121 cases. In the first six months of 1936, 735 cases were reported. However, we have found, from the limited investigations which we have been able to carry out from time to time, that there are existent many more occupational diseases than are reported.

All occupational diseases are due to exposure to industrial health-hazards of one kind or another.

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* From a Radio Talk by DR. HAYHURST, WOSU, July 7, 1936.

Among the chief concepts of a public health department are the investigation of the causes of diseases as well as their extent or prevalence, and the spread of information so that prevention and perhaps complete elimination may follow.

With the enactment of the national Social Security Act, a part of the fund was ear-marked especially to the U. S. Public Health Service for the purpose of extending investigations in industrial hygiene and occupational diseases. Early in 1936, the Ohio State Director of Health, the legislature, and the Governor, promptly approved this Social Security subsidy for Ohio. The portion of the fund for occupational disease work allotted to the Ohio State Department of Health for the balance of the first fiscal year, ending June 30, 1936, was $7,141.66. On July 1, 1936, a new appropriation of $28,000 was made for the next fiscal year. To these amounts are added the present departmental appropriation of $5,200 per annum. These appropriations have permitted the addition to the staff of the Bureau of Occupational Diseases, five technical persons and a secretary-stenographer, along with traveling expenses and a fairly adequate sum for laboratory equipment and supplies.

The Ohio program is divided into immediate and long-range projects. Among the former, the investigation of the more urgent or profound occupational disease cases or complaints is first in order. Here, both labor and employer-organization requests will receive early consideration. Likewise, those of the reporting physician, the given patient, and the given plant or work place, as time and facilities permit. Research and not intrusive investigation is to be the general character of the policy pursued. A preliminary survey will be followed by precision determinations involving medical, chemical, physical and engineering aspects as the indications warrant. In connection with all studies and investigations, another policy will be to proceed without respect to any compensation features. In this matter we call attention to Sect. 1243-3 G.C., Ohio, which reads, in regard to occupational disease reports, "Reports made under this act shall not be evidence of the facts therein stated in any action arising out of the disease therein reported." The purpose, therefore, of the new program is to ascertain the relation between cause and effect, and to make use of this information to prevent further similar mishaps, not only to the given worker, and in the given work place, but among all similar workers in all similar work places.

The long range projects will consist of physical examinations of workers subjected to prominent hazards, such as dusts and certain poisons, infections and physical hazards. Again, surveys, first of preliminary nature, then of more thorough and technical manner will be carried out. The uncertainty of the status of silicosis in the numerous silica-using industries in the state has been selected as the first long-range project. The examination of silica-exposed workers, now or previously employed, including x-ray chest examinations, will be among the initial procedures. A complete equipment for stereoscopic, fluoroscopic, chemical and microscopic determination is to be provided. Not only a newly equipped local laboratory in Columbus, but also a special automobile traveling laboratory with examination quarters are now being set up for this special purpose.

Thus, with the advantage of a good many years of experience in occupational diseases in the State of Ohio, and the selection of proper personnel and the extra-technical facilities made available by the Social Security subsidy, and the wholehearted support of state officials, a new program, backed with enthusiasm and a will to accomplish, is now crystallizing to insure better health of employees in the second largest industrial state in the nation, as reflected in dangerous occupations.

Proceeding through on the theory that all important industrial health-hazards can be eliminated—and we have yet to find one which cannot—we believe that much safer industrial environment can be assured for all who work, and various diseases such as the industrial poisonings, silicosis, and others, can be practically eliminated if not abolished.

Lead Poisoning Reports

FOLLOWING are Dr. Hayhurst's amended forms for the reporting of lead poisoning in Ohio:

COMMENT on the Diagnosis of Lead Poisoning:

1. DEFINITIONS—*Lead*—The element Pb (plumbum), its organic or inorganic compounds or forms, or admixtures thereof or therewith.

Lead Exposure—Any environmental condition or practice that permits of entry of lead into the body.

Portals of Entry for Lead—Refers to the respiratory tract and the digestive tract. Lead may likewise be implanted in the body through direct physical means, by hypodermic injection, or by intravascular administration. With the exception of tetraethyl lead, and related organic compounds, its absorption through the skin or through abraded or lacerated surfaces may be considered negligible.

Lead Ingestion—When lead enters the body by way of the alimentary tract.

Lead Inhalation—When lead in a dust, spray, vapor or volatile form enters the respiratory passageways through breathing effort.

Lead Absorption—When lead enters the body through any portal and is taken into the tissues.

Lead Poisoning or Lead Intoxication—When absorbed lead causes subjective symptoms with objective findings.

Chronic Lead Poisoning—When absorbed lead causes protracted manifestations with or without acute episodes.

Lead Complications—When absorbed lead initiates or aggravates any other disease condition.

Lead Sequela—When a pathologic condition remains as a residum of antecedent lead poisoning.

2. BLUE LINE IN GUM MARGINS. Examine after use of tooth brush. Examine both labial and lingual surfaces. The deposit occurs in the papulae of the gum mucosa so that a fine, stippled appearance may at times be seen when a hand lens is used. Is due to lead in the blood stream combining with sulphur in foods. Occurs in about 20% of cases of leading (absorption of poisoning). The condition is less likely to be found where oral hygiene has been good; also when gingivitis or gum atrophy occur (and these latter may be due to the lead deposit). NOTE: A dark line occasion-

ally occurs after medicinal *bismuth injections*, hence this must be ruled out. Likewise, examine *anal* muco-cutaneous junction for blue line due to same causes as above.

3. BLOOD FINDINGS. *Stippling, polychromatophilia, reticulocytosis, and basophilic aggregation* are apparently but graduations of the same picture, according to technique used. These occur more readily in leading than in other pathological states. They indicate an increased concentration of lead (or its effects) in the blood stream. Although exceptions occur, they usually remain in the blood as long as exposure to lead persists, and tend to decrease upon withdrawal from exposure, even though the case passes into *Lead Poisoning*. They may reappear, or increase, during acute episodes in the course of Lead Poisoning. Their number is taken as a rough measure of the amount of exposure (again, with individual exceptions). Hence, workers on the job who show these red cell changes, even without other evidences of leading, should have blood examinations made frequently (preferably weekly), and advisedly, be removed from exposure on evidence of progressive increase of these cells.

In *Lead Exposure*, hazard may be inferred when stippled cells occur to the extent of about 500 per million red cells (five in 50 average fields), reticulocytosis or basophilic aggregation to 1½ to 2% (of red cells). In general, the larger the granules in stippling, the greater the significance, as indicating excessive absorption of lead.

When *Lead Poisoning* is definitely established, fluctuating retrograde changes occur in the whole blood picture. The more constant findings are: (1) RBC—secondary anemic, disturbed coloring properties, variations in size and shape, and (2) WBC—slight or moderate leucocytosis (increased with infected gums, etc.), slight relative lymphocytosis (changing to lymphocytopenia during acute exacerbations), and mononucleosis.

4. Reports of *Analytical Findings* of *lead in the urine, feces and blood* are of doubtful value unless accuracy and delicacy of testing methods are assured, and the patient is under control as to extraneous sources of lead (food, water, tobacco leadfoil, cosmetics, etc.). Also, the test observations must be made over days and preferably weeks to get a true picture. "Normal lead" probably exists in the amounts of 0.05 to 0.10 mgm. per liter of urine, and to a somewhat higher proportion in the blood.

5. SINGLE *or occasional negative laboratory findings do not rule out Lead Poisoning*, but positive findings are quite significent. Also, *deferred laboratory tests* (months after exposure) are usually negative as regards all specific or "characteristic" findings for lead. Here, some have found them to return for a brief period during de-leading treatments.

6. BLOOD PRESSURE is subject to wide variations under the influence of lead. Hypotension is more common in early stages, hypertension in longer standing cases.

7. Therefore, the *Diagnosis of Lead Poisoning* is based upon the weight of certain evidences: (1) Definite *exposure* to inhaling or ingesting lead or its compounds in amounts equal to at least one and one-half to two milligrams per day, over two or more months' time—less time if greater concentration—and in the form of dust, sprays, molten fumes; (2) *objective findings* of lead absorption (blue line, blood changes, etc.), neuro-vascular involvements (motor weakness in most used parts, pallor, tension disturbances), and nutritional depletion (especially in chronic cases); and (3) the presence of a number of the *subjective phenomena* listed,—the more acute cases showing those toward the tops of the two columns. To be occupational, industrial exposure must be established.

Supplementary Evidence in the Diagnosis of Lead Poisoning

Patient's Name..... Address.....

Physician's Name..... Address.....

* * *

1. Has this patient had previous attacks of lead poisoning? Give approximate date(s).
2. What were the onset symptoms in the present attack?
3. Was disability such that work was stopped? If so, when?

Check or Comment on the Following Items

I. Objective Findings (i.e., the physicians observations)

Pallor (especially circumoral)	Facies "cachexiae"
Blue line in gum margins	Gums, teeth (condition of)
Blue line at anal margin	
Blood Findings—	
Stippling (basophilia of red cells) or	RBC—variations in size, shape
Polychromatophilia or Reticulocytosis or	RBC—resistance to hemolysis and rouleaux formation
Basophilic aggregation (%)	Lymphocytes (%)
Hemoglobin (%)	Monocytes (%)
RBC count(s)	WBC count(s)
Recent or recurrent retrograde blood changes?	
Blood Pressure Findings	Pulse (at rest)
Arteriosclerosis	Hematophorphyrinuria

Finger strength (extensor, flexor actions)
Hand-gripping power (condition of): R..... L.....
Wrist extensors (any actual wrist drop?): R..... L. ..... Any edema?

Tremor	Incoordination	Ataxia
Reflexes: Patellar		Ocular
Vision: R.....	L.....	Recent disturbances
Hearing: R.....	L.....	Recent disturbances
Malnutrition	Emaciation	Premature ageing
Gouty signs	Evidence of nephritis	
Skin (acne, etc.)	Weight variations	Mental disturbances
Luetic Test(s)		

II. Subjective Findings (i.e., the patient's complaints)

Morning anorexia	Metallic (or sweet) taste
Nausea or vomiting	Distress in abdomen
Obstinate constipation	Abdominal colic attacks
Headache	Insomnia
Rheumatic pains	Muscle cramps
Dizziness	Mental depression

Weakness (hand grips; use of wrists, legs, foot, etc.)

General weakness	Syncopal attacks
Disturbed vision: R.....	L. .....
Deafness: R.....	L. .....
Weight: Normal	At present

III. Any Further Findings or Comments

.....

.....

Physician's Signature .....

Date.....

The Painful Shoulder

By E. B. MUMFORD, M.D., F.A.C.S.
Indianapolis

PAINFUL shoulders are common problems in the practice of medicine, presenting to the physician or the surgeon the problems of diagnosis and treatment and for the patient the elements of a painful disability and a long convalescence.

In the anatomy of the shoulder region, the shoulder joint, classified as of the enarthrodial or ball and socket type, is dependent entirely upon the muscles and the tendons of the shoulder group for its stability and for its wide excursion of motion in practically all directions and through all planes. The ligaments between the scapula and humerus are blended with the capsule which is thereby strengthened, but neither the ligaments nor the capsule hold the two bones in contact but merely limit the range of motion of the joint.

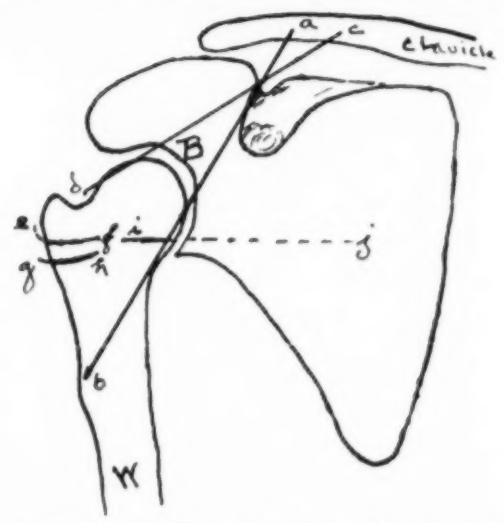
These factors are not only known from anatomical study but are readily demonstrated in the paralytic shoulder, in which condition the humerus drops from the acromion and dislocation can be esily produced. The bursal sacs are numerous (eight described in Gray's anatomy) and may not only be traumatized but also are subject to the same reaction to toxin and bacteria as other bursae. Although the subacromial (sub-deltoid) is the largest and perhaps best known on account of its association with calcareous deposits, the smaller sacs all play a role in the "painful shoulders," especially that bursa between the coracoid process and the capsule.

Perhaps the most interesting and at the same time least understood aspect of the shoulder joint is that relative to its physiological function or to its kinetics. The motion of the arm as a whole through the shoulder joint is one of leverage (third type) with the scapula (glenoid fossa) the base, the hand and forearm the weight and a portion of the shoulder muscle group the power. In any leverage action the first essential is that the fulcrum be fixed. The scapula, with its glenoid cavity, is a mobile bony structure only at the sternal end of the clavicle, the clavicle being only a buttress. In order that the scapula become fixed before abduction can be made of the humerus (arm) there takes place a well coordinated but intricate contraction of all the muscles which hold the scapula to the trunk. These muscles contract and relax in varying degrees and even perhaps in different fibers of the same muscle, as the plane or axis of the glenoid cavity is changed to make possible the different degrees of abduction, as abduction can only be made from the straight dependent position to ninety degrees, further motion in the joint proper being prevented by the bony structures and by the capsule of the joint with its inherent ligaments. For abduction beyond ninety degrees it is necessary that the glenoid cavity be tilted. The axis of the glenoid cavity must also be changed in making full abduction as well as in reaching forward or backward. This shifting of the scapula is easly demonstrated by placing the hand upon this bone.

The second essential factor in the leverage action is that the lever (humerus) be held firmly against the fulcrum (glenoid fossa). It has been stated that the capsule and the ligament do not hold the humerus in contact with the scapula but that the head of the humerus has a rather wide excursion within the glenoid from a low position to one higher and close against the acromion process. In order to give this essential constant contact between humerus and scapula the subcapularis swings from within across the neck of the humerus to meet the tends of the infra-spinatus and the teres minor comes from the outer side to form a sling or band which holds the humerus against the scapula, a position which can change as these muscles contract in varying degree.

The exact mechanism in abduction is in dispute. Stevens has given a very logical explanation in his discussion of the shoulder in which he shows that it is impossible for the deltoid (unless selected portions of the muscle can act independently of the other portion) to start the motion of abduction as the resolved line of pull of the muscle from its origin on the scapula to its insertion on the deltoid falls inside of the fulcrum and would cause adduction rather than abduction of the arm. Undoubtedly the supra-spinatus is an abductor and its purpose that of starting abduction and bringing the arm into that degree of abduction in which the line of pull of the deltoid falls outside the fulcrum so that this muscle can act as an abductor.

Leverage Diagram of Shoulder
W —Weight (arm)
B —Base (glenoid cavity of scapula)
a-b—Force line of deltoid
c-d—Force line of supra-
e-f —(Sling) Force line of infraspinatus
g-h—(Sling) Force line of teres minor
i-j —(Sling) Force line of subscapularis



When abduction through the deltoid has reached about 90°, further motion in this direction cannot be made in the glenoid cavity as the tuberosity of the humerus impinges against the acromion and the inferior portion of the capsule becomes taut. Further abduction is made possible by changing the direction of the plane of the glenoid cavity through shifting the position of the scapula as a whole upon the trunk. This brings into play a relaxation of certain muscles and contraction of other muscles which had previously immobilized the scapula as a fulcrum. In the last phase of abduction the biceps play a part. A complicated, smooth working and delicate coordinated mechanism which is almost beyond conception by the human mind — another evidence of a marvelous evolution from lower animal life or of a finished product by a Master Creator whose original plan was perfect and has not been changed!

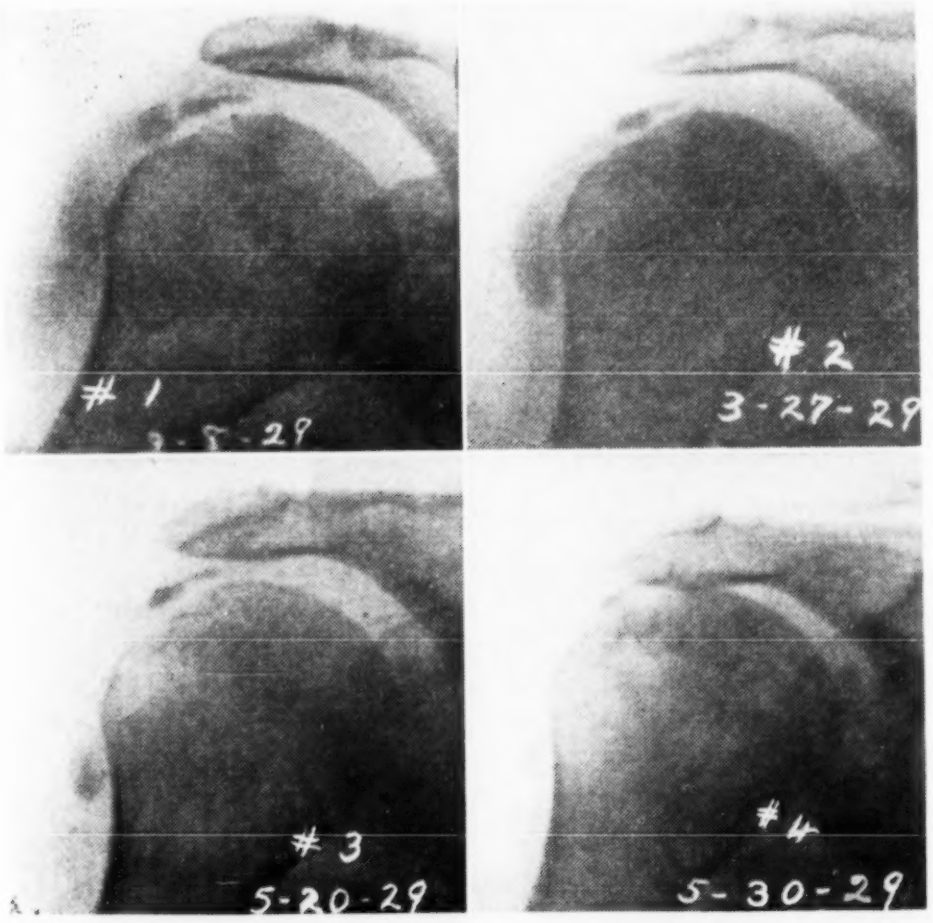
The relation of the tendon of the supra-spinatus to the under surface of the tip of the acromion is of importance in one type of shoulder lesion. As the arm is abducted this tendon is brought into contact with the acromion and eventually glides entirely beneath it. As a result of this continuous pressure the tendon undergoes changes through attrition which lead to a thinning out or even a fraying of the tendon. This chronological

change and its role in the painful shoulder will be discussed later. The long head of the biceps muscle, through its peculiar relation to the humerus and its attachment to the rim of the glenoid cavity is subjected to a frequent trauma somewhat similar to that observed in the tendon of the supra-spinatus muscle.

Determining the exact causative factor and visualizing the pathological changes in a "painful shoulder" is often a most difficult problem requiring a careful clinical examination supplemented by x-ray study. Replogle, in a study of 150 consecutive shoulder cases occurring in the Cambria Steel Works, found 26 different types of lesions.

A classification of the factors which give rise to pain in and about the shoulder joint suggests five groups of cases, in each of which the form of treatment and the prognosis vary greatly:

Group 1. Those associated with trauma either direct, as contusion to the shoulder area, or in-



Sub-acromial Bursitis with Calcareous Deposit — Treated with Diathermy.

direct, as found in injuries to the fore-arm or to the hand, those following severe sudden trauma as fractures and dislocations and those due to repeated slight traumas over a long period giving rise to degenerative changes in the tendons.

Group 2. Those cases which can be termed as peri-arthritis and follow some focal infection in teeth or tonsils and in which the bursal sacs are perhaps alone involved.

Group 3. Those painful shoulders in which the x-ray reveals a calcareous deposit in or about the sub-acrominal bursal sac.

Group 4. This group includes those cases in which there is a definite infection involving the articulating cartilage. This infection may be due to the pyogenic organisms or to the tubercle bacillus.

Group 5. This group includes those cases in which the etiological factor is varied greatly. Lesions involving the phrenic nerve, the pleura, lung, mediastinal glands and the abdomen as well as malignant tumors may have pain referred to the shoulder.

Those cases following fractures and dislocations are readily diagnosed while those falling in the fifth group are uncommon and shall not be discussed.

Group I. The traumatic shoulder, other than those of fractures and dislocations, is perhaps the most common type of "painful shoulder" in which the diagnosis as to pathological changes is confusing and uncertain. The history may be one of a definite accident as a fall upon the fully extended arm or upon the elbow or upon the partially abducted arm or it may be merely that of a reaching motion without force. The onset of the pain is usually sudden and sharp and associated with a considerable loss of function. The pain may be referred to the tip of the shoulder or down the arm in the region of the attachment of the deltoid muscle. Abduction is possible only to about forty degrees and both internal and external rotation are restricted, the patient being unable to get the hand to the top of the head or to the back of the body. There is a constant ache and sleep is disturbed as the patient can not find a position which is not painful for the shoulder. Rarely does one see this type of "painful shoulder" in a person under 40 years of age.

Upon examination one finds the patient carrying the arm at the side with every evidence of pain. Removal of the clothing is difficult. In the early period one does not find any atrophy of the muscles but later there may be noted a marked change in the supra-spinatus space. The deltoid as a rule shows but little or not any change in size. Swelling is not marked and the tenderness is spotty, the area over the coracoid-humeral bursa being most constant with that of the deltoid tubercule and the quadrilateral space less frequent in this respect. A tender point beneath the tip of the acromion is not uncommon.

If the supra-spinatus tendon has been torn Codman gives 18 points in diagnosis as follows: occupation of labor, age over 40, no previous symptoms, adequate injury, immediate sharp brief pain, severe pain several hours later, loss of power in elevation, negative x-ray, little or no restriction of motion of shoulder when stooping, faulty scapulo-humeral rhythm, tender spot, a sulcus over the tendon, an eminence at the insertion of the tendon of the supra-spinatus which causes a jog and a wince and a soft crepitus when the tuberosity glides under the acromion as the arm is abducted and with the same symptoms reappearing as the arm is lowered from the abducted position.

The pathological changes which occur in this lesion are not always clear as to which tendon is involved. Mayer, in his anatomical studies, found the long head of the biceps in adults to be frequently attenuated or frayed or ruptured and his findings have been confirmed by other anatomists. As a result of his studies he made the following conclusions: 1. Uncomplicated partial or complete, spontaneous, forward dislocation of the supra-tuberosital and inter-tuberosital portions of the tendon of the long head of the biceps, on or over the tuberosity as far as the tendon of the subscapularis, is common in laborers past middle life. 2. This dislocation may be due to gradual stretching, detachment or destruction of the capsule proximal to and in the region of the lesser tuberosity. 3. Wear of the tendon along its dorsal margin may occur in otherwise normal joints, but wear throughout its width probably is

frequently due to contact with the lesser tuberosity and the acromion in the course of destruction of the dorsal portion of the articular capsule, unaccompanied by dislocation. 4. After division by attrition, the tendon usually obtains a secondary attachment to the humeral diaphysis immediately distal to the lesser tuberosity, or to the floor of the sulcus in this region, or still more infrequently to both walls of the sulcus, a portion of the frayed tendon being drawn to each side by tendon sheath in the region of reflexion of the latter.

Codman, in an analysis of 970 cases of shoulder lesions, found the supra-spinatus involved in 234 and the biceps in only 18, and therefore considers the supra-spinatus as the chief offender in these painful conditions.

If a very definite diagnosis of a complete rupture of the supra-spinatus or biceps tendon can be made, an immediate suture of the tear through open operation will no doubt lessen the convalescence and reduce the impairment, but this operation is not always a simple one as the tendon may be retracted beneath the acromion to a considerable distance and its suture to the shred of tendon remaining on the head of the humerus difficult. The vast majority of these cases come to the surgeon at a late date and the prognosis following simple treatment is sufficiently good to question the advisability of an operation at this time.

Associated with any tear or split of the tendon there is without doubt a marked reaction of a mild inflammatory type with an infiltration of the bursal sacs and other adjacent soft tissues by blood or lymph. It is this infiltration which gives rise to the late symptoms of loss of function and pain on certain motions through the formation of adhesions. This premise can be assumed for when the shoulder is manipulated under anesthesia one feels and hears the breaking down of adhesions and later almost complete return of function of the shoulder without pain can be expected.

The non-operative treatment of the traumatic shoulder in which adhesions are the chief offending factors may be the slow method of Brickner in which the arm is gradually brought into full abduction. The head of the bed is raised about six inches and with the arm fastened to the head board in as full abduction as is comfortable to the patient and with pillows to support the arm, further abduction and at the same time stretching of the adhesions takes place during sleep as the patient gradually slides down towards the foot of the bed with the arm still fixed to the top of the bed. Watkins suggested a more rapid attack upon the adhesions by bringing the arm into full abduction under anesthesia and maintaining this position for two or three days by means of a lateral splint. Following both methods the patient is urged during the day to use the arm to the fullest extent in order to maintain all the gain made through the method of treatment. Massage, heat and liniments play a minor but perhaps an important role in the recovery.

With any plan of treatment the patient should be advised that recovery will be slow and that the pain will persist for many weeks but one can also assure him that the prognosis is good for entire relief of pain and an almost complete return of function.

GROUP II: The second group which has been termed peri-arthritis is similar to the first group in that the condition is one of a painful shoulder with a loss of function most marked in the ranges of abduction and internal and external rotation. The etilogical factor is some fomal infection with an inflammatory reaction in the bursal sacs about the shoulder joint as the result of either toxins or of a mild grade of bacteraemia. Swelling and tenderness over the front of the shoulder is common. In this group every effort should be made to locate the area of infection and although the teeth, sinus and tonsils are frequent offenders, one must not overlook that old criminal, the prostate gland.

The treatment in the initial acute stage should be heat and rest. Morphine may be required for the pain which may be most severe. Aspiration may give a cloudy fluid. During this period a vaccine may be given and all avenues of elimination kept at work. After the acute stage has subsided and the swelling and general tenderness has disappeared the treatment is similar to that of the traumatic group and the prognosis may be estimated along the same lines.

GROUP III: The diagnosis of the painful shoulder due to calcareous deposits in the subacromial bursa is dependent upon the findings in the x-ray. These cases have no relation to labor, being found in men of all vocations and also not infrequently in women who are not doing heavy work. The onset is usually sudden with an acute pain so severe as to produce complete disability. In some instances the condition comes on slowly but there is, as a rule, some single movement which brings on an acute exacerbation of pain. Atrophy of the muscles is not present and swelling is uncommon. Tenderness over the head of the humerus is marked and the pain may radiate down the arm and even into the forearm. Morphine may be required to relieve the distress.

The x-ray shows the characteristic shadow of a calcareous deposit just below or beneath the tip of the acromion. The composition of this deposit has been variously interpreted and most investigators upon operation have found the deposit lying beneath the bursal sac rather than in it. The interesting feature is that this deposit has existed for some time before the onset of the pain and has often been found in shoulders free of symptoms. The writer has case records of thirty-six of this group and is of the opinion that the deposit in some manner causes an irritation, either through increase in size or through some particular trauma. to the bursal sac.

The treatment of these cases is either operative or with diathermy. Many instances have been reported in which the deposit has disappeared without any treatment. In the 36 cases of the writer, complete relief of all symptoms and in most cases complete disappearance of the deposit have followed the use of diathermy. Upon relief of pain these cases do not have the formation of adhesions as found in the first and second groups and the return of full use of the arm has followed without any special treatment directed to this feature of the disability. Relief of pain is obtained within four to ten days and the disappearance of the deposit takes place within three weeks.

GROUPS IV and V: It is not the purpose of this paper to discuss the fourth and fifth groups. The cases of purulent arthritis of the shoulder are un-

common and as a rule follow some definite direct infection such as puncture wounds or compound fractures. One case has been observed following an acute epiphysitis in a child. Tuberculosis is rare in the shoulder. The joint is usually overlooked by the gonococcus. The cases in which the pain is a referred one require a most careful and widespread examination and are the types in which the diagnosis is so often undetermined. The history should be most painstaking in character and will be the lead in locating the distant pathological condition.

Gas Gangrene

By BATTLE MALONE, M. D.,
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GAS GANGRENE is a condition which is of importance to every one who has to do with the care of wounds.* Before the War we knew and spoke of the Bacillus aerogenes capsulatus and Bacillus oedematis maligni, but were truly ignorant of the manner in which they worked and are now conscious of our very feeble efforts of combating their dire affects on the tissues of the human body.

From the great number of cases, which are being reported with increasing frequence, we must conclude that the possibility of its occurrence is always present and there is little that we can do to lessen this possibility, for it is essentially a disease due to pollution of the soil, and the older and more thickly settled a country becomes the greater is the pollution of the soil. Wounds which are contaminated with dirt in stable lots, in gardens or on public highways, are particularly susceptible to infection with anerobic germs. Urban Maes calls our attention also to the danger of infection through contamination from woolen clothing and bed clothing. There are many anerobic micro-organisms, but most of them are not pathogenic.

During the War, before America entered, the problems of gas gangrene had been pretty definitely worked out by the Pasteur Institute, and it was their conclusion that there were but three micro-organisms to be feared. These were: The perfringens, which is the same as the B. aerogenes capsulatus or, as we in America now know it, Bacillus Welchii, so named for its discoverer; the Vibrion septique (B. oedamatis maligni) and the B. oedematiens (B. bellonensis). Other anerobic organisms found in cases of gas gangrene are thought to be present only by symbiosis. French surgeons by concentrating their attention on these three bacilli were much more successful in reducing the mortality of the disease.

In this country it seems to be a definite, established fact that the B. Welchii is the prevailing or greatly predominant infection organism. Elliott and Easton, in reporting 17 cases, state that the B. Welchii was the causative factor in all. In 1931 a committee from the New York Hospital for Bone and Joint Diseases concluded that all gas infections in this country were caused by the B. Welchii. The reason for stressing this fact will be brought out later when discussing the use of sera in the treatment.

While it is possible to have gas gangrene in any wound (cases have been reported where it developed after a simple hypodermic injection) there are certain types in which we fear it most —cuts, laceration, and especially in compound fractures incurred around barns or stable lots, in gardens, or any fertilized plots, or on our highways, are particularly liable to be contaminated by anaerobic micro-organisms. And this is equally true of gunshot wounds through woolen clothing.

It is in such wounds, especially where there is much laceration of muscle, that gas gangrene develops. It manifests itself in a short while, usually in the first 24 hours, rarely later than 36. The signs and symptoms are easily recognized. There is a marked oedema, rapidly increasing, due to the formation of gas in the muscles, extending rapidly up the fascial planes and escaping from the wound, giving definite emphysema on palpation. The peculiar sickening, sweetish, foul odor is unmistakable. The muscles affected have the appearance of parboiled beef. The enormous tension of the imprisoned gas quickly causes interference with the circulation. Thrombosis of the main vessels and massive gangrene quickly occur. Constitutional symptoms of profound toxemia, and it is purely a toxemia, develop rapidly. They are the symptoms of severe surgical shock—rapid pulse, cold, clammy sweat, extreme restlessness plus high temperature. If not treated promptly the gangrene extends rapidly and death comes quickly.

The development of this disease can be prevented by prompt and proper treatment, which consists of the free use of iodine in and around the wound and careful and complete removal by dissection of all contaminated and dead tissue—what the French call debridement. If there is much laceration of muscle it is better not to attempt immediate closure. If the wound is not seen within the first or eight hours it is better to leave it open anyway. Smears and cultures should be made, and if the wound is open should be repeated frequently. In the open wound there should be placed Dakin tubes and the Carrel-Dakin technique carried out scrupulously.

We hear considerable criticism of Dakin fluid, and when I hear it I think that the critic has been using something called Dakin, which is not. Dakin solution, properly made, when titrated should contain between .45 and .5 chlorine. This, when properly used, I know cleanses a wound and keeps it clean. Wounds which have been kept open can be closed as soon as one knows they are free of infection. Such care as has been outlined is the best way to prevent gas gangrene. After it has developed, as sometimes happens, it is limited to a certain muscle or group of muscles and massive gangrene has not occurred, the limb may be saved by dissecting out completely the affected tissues. One can easily tell by the quick retraction of the muscle fiber when healthy muscle is reached. When such treatment is employed the Carrel-Dakin technique should be immediately utilized and cultures made frequently and appropriate sera administered. If massive gangrene has occurred, immediate amputation is indicated. This should be done by the guillotine method. I have found that in doing a guillotine amputation it is possible to leave a cuff of skin which is folded back and can be used to cover the end of the stump, when the wound is free of infection. Immediately the amputation is completed the Carrel-

* Presented at the Thirty-Ninth Annual Meeting of the Association of Surgeons of the Southern Railway System, June 16, 17 and 18, 1936.

Dakin treatment should be begun and the proper serum used. A little later more will be said about the sera. I am happy to say that by carrying out such measures as have been outlined, I have not had since the War a case of gas gangrene develop in a single case of my own. I am also proud of the fact that I have had no mortality in the cases which have been referred to me. The mortality is however, high. At home, at the Methodist Hospital, the mortality is 33⅓%; at the General 36.7%; at St. Joseph's 25%, and at the Baptist 38%. Reports from other locations show there is a mortality of from 30 to 50%. Following the Tupelo disaster last winter six cases occurred among the victims brought to the General Hospital, with three deaths.

Now let us consider the use of anti-gas gangrene sera, both as used as prophylaxis and curatively. I had the pleasant experience of working at the Rockefeller Institute during the War when Bull was developing his antitoxin against the Welch bacillus. He proved very positively that gas gangrene is a toxemia. Then one may ask why it should not be treated successfully by a proper antitoxin. Occasionally we have a report that this has been done. Dearing, of the Navy, reports a case of a severe gas bacillus infection in a compound fracture of the ankle cured by injecting into the vein six doses, each of 20,000 units of serum. Joseph reports two cases of fracture of the pelvis with gas bacillus infection (Welch bacillus) cured by the use of serum. In such cases as the last, where amputation cannot be done, one must rely on the curative effects of the serum, but when massive gangrene has developed in a limb, of course amputation must be done.

I am firmly convinced that the administration of the serum in therapeutic doses is of definite help. During the War, while with the French Army, I was fortunate in having the opportunity to work in Du Vall's Hospital, where Vaucher did such remarkable work with gas gangrene. The French were convinced that a polyvalent serum, potent against the three anerobes mentioned above as pathogenic for gas gangrene, could not be produced and so they had three sera, one specific for perfringens, one for Vibrion septique and one for B. oedematiens. Immediately upon admission to the hospital of a convoy of wounded, Vaucher would make smears and cultures from every wound, and where contamination by a gas bacillus was found the appropriate serum would be administered. By following out this plan in the later phases of the War the mortality was reduced 35%, by far the lowest during the War or since. Keen states that in this hospital polyvalent sera were used, but this is a mistake. The procedure was as related above.

Those who manufacture bacteriological products in this country are marketing polyvalent sera, supposed to be potent against all of the gas bacilli as well as against tetanus. It seems to me that it would be better if we would follow the example of De Vall and Vaucher and have appropriate sera to be given in accordance with the type of bacilli found in each case. My own practice has been to rely on the serum potent against B. Welchii, since that is the only infecting agent in cases with which I have had to deal. If a case should occur where the Vibron septique or the B. oedematiens was found we would at present have to depend on a mixed serum.

Now as to the value of the prophylactic serum. For a number of years manufacturers have supplied a serum containing 1500 units of antitetanic serum and 1000 units of Welch bacillus and Vibrion septique sera. So many cases of gas gangrene developed after this so-called prophylactic dose was given that the number of units of the Welchii and Vibrion septique was doubled. A great many men doing industrial surgery have used this combined prophylactic serum. I would not say that it never prevented gas gangrene, but too many cases have occurred following its administration to permit me to consider it in any way efficacious. A member of a group of orthopedists at home tells me that they have had five cases developed after its use. At the General Hospital there were seven cases. Tilford reports two cases developing after its administration and one of these three doses was given. Finsilver reports a case where there was a recurrence three times in spite of phophylactic doses.

I have seen in consulation two cases, in one of which the ordinary dose was given and gangrene developed in 24 hours. In the other, not the ordinary dose, but one of 10,000 units of a bivalent serum (against B. oedematiens and Vibrion septique) was given and 24 hours later there was definite gangrene. Bacilli Welchii were found and after amputation therapeutic doses of 10,000 units of straight anti-Welchii given, and the patient recovered. With this evidence before us one cannot be blamed for having little faith in the product which has been furnished. If we want to use a prophylactic serum it would be better to follow the example set by DuVall and Vaucher until some better method has proved its usefulness.

Sometimes the condition of the patient is such that prompt debridement cannot be done. Frequently it may be necessary to treat the patient for shock before any operative procedure may be done. Or it may be impossible to get the patient to the hospital promptly. In such cases, in my opinion the proper course to pursue would be to give the straight antitetanic serum, and at the same time give 5.000 to 10 000 units of Welchii bacillus antitoxin. Then as soon as possible proceed with the surgical treatment.

What has been said here applies chiefly to gangrene of the extremities.

A good many cases have been reported of gangrene of the abdominal wall, following operation on the intestines or gall bladder, or the removal of gangrenous appendices. Hugh Gamble made a very interesting report of 12 such cases at the last meeting of the Southern Surgical Association. He suggests that the proper method of preventing such a complication is to leave the abdominal wound open.

In recent years attention has been called to still another location for the invasion of gas bacilli, namely in the uterus. Numerous reports are found in the literature. Infections usually occur at parturition but may happen following abortions. From the Maternity Department at the Memphis General Hospital I find recorded nine such cases occurring since 1931. There were five deaths and four recovered, and here I think we find a significant fact, certainly one which proves to me the efficacy of the antitoxin: of the five deaths only one received the perfringens serum; of the four who recovered all four were

given therapeutic doses. In seven of the cases the perfringen was found in smears and cultures:

CONCLUSIONS:

1. Gas gangrene can be prevented by prompt removal of all contaminated material and tissue from a wound.
2. Gas bacillus antitoxins are of great value, but only as aids to proper surgery.
3. The prophylactic serum supplied by manufacturers has not proved its efficacy. A larger dose of a B. Welchii antitoxin is to be preferred.

National Advisory Committee

On Toxic Dusts and Gases

THE American Standards Association is the recognized national clearing house for the establishment of uniform safety codes. Approximately 40 such safety codes have been approved to date and some 20 more are in the course of preparation. These codes have been widely used by state regulatory bodies as the basis of state regulations, by insurance companies in recommendations to their assured, and by individual industrial concerns as plant rules for the removal of hazards in conducting industrial processes.

The development of standards in the field of occupational diseases has recently been undertaken, chiefly through the work of the committee charged with the responsibility of preparing standard codes for exhaust systems. It was through this committee that the initial recommendation for the establishment of a National Advisory Committee on Toxic Dusts and Gases was received by the American Standards Association. This code committee felt that it was inadvisable to proceed with the development of specifications covering the design, operation, and maintenance of exhaust systems for use in any particular industrial operation unless authoritative information could be obtained concerning the threshold limits of toxicity of the particular dust or gas which the exhaust system is designed to remove from the atmosphere being breathed by workers. It was also recognized that there are various opinions as to what the threshold limits are for various dusts, gases and fumes, and a correlation of these opinions through some central organization is highly desirable.

It is not contemplated that the Advisory Committee will be called upon to establish threshold limits for all toxic dusts and gases, but it will serve as an advisory committee to the various code committees established under the procedure of the American Standards Association which are preparing standards covering particular occupational diseases.

It will be called upon to advise such committees along the following lines:

1. Determine whether or not sufficient technical data exists to permit the committee to proceed with its work.
2. Establish threshold limits for particular toxic dusts, gases, and fumes, for the use of code committees.
3. Assist code committees in establishing research programs when necessary.

Many organizations have expressed the need for a National Advisory Committee of this kind, and recognizing the clearing house functions of the American Standards Association, have encouraged and endorsed the establishment of such a committee.

Representatives of some 25 such national groups have cooperated in planning the program of codes for the prevention of occupational diseases, including the setting up of the National Advisory Committee on Toxic Dusts and Gases. Among these is the U. S. Public Health Service, and Dr. R. R. Sayers has accepted the Chairmanship of this Advisory Committee.

The committee will be composed of leading toxicologists, pathologists, industrial physicians, industrial and chemical engineers, and other experts who have achieved national recognition in this field.

Osteomyelitis

Questionable Traumatic Osteomyelitis of the Phalanges

By J. ROBERTSON KNOWLES, M.D.,
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TO DATE there has been a tendency for the medical man to attribute most cases of osteomyelitis, especially of the phalanges, to some remote injury. This tendency has caused the insurance companies and industry to pay a number of unjust claims.

I believe we are wrong when we state that an osteomyelitis of any phalanges is due to direct trauma, and I do not believe we can state this any more than we can say that an appendicitis is due to direct trauma.

We must remember that acute infections of bones are caused by direct or indirect invasion of pyogenic organisms, and it is these organisms which produce the periostosis, osteitis or osteomyelitis, (not trauma); and we must remember that these organisms either must be in the system at the time, or else they must gain entry through some abrasion in the skin at the site of contact. We must also remember that our low grade infections usually occur in bone as the result of an infection within the body.

Although we see many palmar abscesses with cellulitis and lymphangitis (the typical "ham-hand") yet in these badly infected cases we very rarely see an osteomyelitis of the metacarpal bones or any of the phalanges.

In going over the literature on the subject I have been unable to find any record to date where there has been an osteomyelitis of the proximal or middle phalanges of any finger, without having an osteomyelitis of the distal phalanx as well.

This is easily explained when you consider the anatomy of the finger and note that branches of the blood supply reach the tip and the last joint of the finger, but do not supply the diaphysis; and this I believe causes the early necrosis of the shaft which we see in a true osteomyelitis of the phalanges.

For this reason I believe that trauma to the fingers is only a predisposing factor in these cases, the trauma lowering the local resistance of the part and not directly traumatizing it as appears to be the accepted theory today.

The History of Industrial Medicine and Occupational Diseases

—An Historical Outline of Occupational Diseases, Industrial Hygiene and Surgery, Traced from Early Antiquity to the Great Depression of the Twentieth Century—

by

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Metals, Gases, Solvents and Explosives

THE chemical age dawned in the latter part of the nineteenth century, when synthetic and analytic chemistry developed. Useful discoveries in these fields have brought about social and industrial changes which are almost revolutionary in their scope, but which entail a train of industrial and domestic hazards of corresponding significance. The cracking of petroleum, fractional distillation in wood and asphalt, the splitting of coal tar molecules, and the discovery of many new elements are all fairly recent accomplishments which have contributed to man's usefulness. They gave to the arts the dyes, explosives, solvents, lubricants, pigments, and modern building materials; to medicine, antiseptics, drugs, perfumes, pigments, and radio-active substances; and to domestic life, fabrics, fuel, floor coverings, artificial leathers, insecticides, cleaning compounds, etc. Every new process, improvement, or discovery, whether it be chrome plating, making of luminous watch dials, aeroplane wing doping, tetraethyl lead, or T.N.T. explosives, brings a new occupational disease.

The hazards from chemical agents, such as metals, fumes, dusts, and solvents, constitute the bulk of the danger of occupational diseases. It is stated that in four out of five trades potential hazards exist. Hence a discussion of the special elements, metals and gases, with the scientific investigations that contribute to the study of industrial diseases, will not be out of place here. Let us note, too, that besides the individual results of various industrial poisons, W. H. Rand, U. S., in 1920 reviewed composite industrial poisons and quoted Buergi to the effect that the variable effects of a poison depended on several factors; two substances mixed may yield a new chemical body, one may be modified by the other, the penetrability of the cell membranes may be affected by the other, the saturation of the cell with one substance may make it more or less absorptive of another. In this chapter we shall consider the effects of the separate poisons.

Arsenic

PARIS GREEN was discovered in 1712 by Russ and Satler, in Schweinfurth, Bavaria. In an attempt to manufacture Paris Green in 1742 a Swede by the name of K. W. Scheele discovered Scheele's Green. These arsenical compounds, which are used extensively as insecticides and pigments, are the cause of much of the poisoning in the arts. It is stated that Paris in 1825 witnessed some cases of arsenical cancer among industrial workers in tin and copper. Credit must be given to Jonathan Hutchinson who, in 1887, first called attention to this fact.

In 1836 James Marsh first introduced his test for arsenic, and eruptions of the skin as a result of handling arsenic were first mentioned in 1857 by Devergie. Two Germans, Harting and Hesse, in 1878, asserted that an unusual number of carcinoma of the lung was found among miners of the Schnesberg cobalt arsenide mines in Saxon, Switzerland, and linked this fact up with the arsenic. J. J. Putnam, of Boston, in 1890 called attention to the danger of arsenical poisoning from colors in wall paper, especially in damp houses. He also found arsenic in the urine of patients. Paul Brouradel, of Paris, as early as 1897 found arsenic in the hair and nails of patients who had died from arsenic poisoning.

Evidence of the injurious effect of arsenic as an industrial poison, especially among Paris Green workers, was pointed out by Dr. T. M. Legge, of London, in 1900, while J. Glaister, of Edinburgh, in 1908 described the symptomatology of arseniuruetted hydrogen and its toxic effect on liver and blood. The same author in 1910 showed the dangers of decomposing ferro-silicon and stated that it should not be transported in the bottoms of ships. A. Bayet and . Slosse, of Belgium, claimed in 1919 that the skin lesions among briquette workers was not due to the tarry substance, as formerly thought, but rather to the arsenic contained in the tar.

In 1920 Ravant employed sodium thiosulphate intravenously as an antidote for arsenic poisoning.

Cadmium

THE origin of the name cadmium is connected with the making of bronze. That this earth-like substance when melted with copper gave a yellow alloy was known by Aristotle in 320 B. C. It was named cadmia by Dioscorides and Pliny in the first centuries of the Christian era. It was believed to be zinc metal until 1817, when Stromeyer, of Gottingen, discovered it to be a distinct element. Sovet, a Belgian, in 1858 first reported cadmium

poisoning. W. Marme, a German, in 1867 published a paper on the toxic effects of cadmium as shown by animal experimentation.

C. L. Alsberg and E. W. Schwartze, in 1919, observed cadmium from a pharmacologic standpoint and noted emesis as a toxic symptom.

Cadmium is obtained from zinc ores; the greatest hazards are due to fumes and dust. It is used extensively in cadmium plating.

In 1932, Leon Prodon, of the Harvard School of Public Health, reviewed the entire literature, and the toxicity of cadmium, and proved that it is due to inhalation of fumes and dust.

Cobalt

HOFFMAN, in his *Mettalurgia Morbifera*, states "that the men employed in digging and manufacturing cobalt at Kuttenberg in Bohemia are affected with vomiting, syncope, anxiety, cardialqeai, difficulty with breathing, sense of suffocation, tremors, etc., and that they appear like living skeletons."

Chrome

THIS element was discovered by Vanquelin in 1797 and has been extensively used since. As early as 1827 the effects of chromates on workers was published in a work by a Glasgow physician named Cumming. He showed that the chrome holes in the hands and arms of workers were due to the action of chromates.

As early as 1851 Becoust and Chevalier, of France, studied the effects of bichromate of potash on the skin, and Heathcote, a Britisher, continued those studies in 1854, describing its ulcerating effect. A. L. Delpech in 1869 showed that not only the bichromates but the chromates as well could cause ulcerations.

J. B. Hilairet in 1869 investigated the diseases of chromium manufacturers. To estimate the quantitative chromic acid content in air sampling in chrome-plating industries, the Dubosq Colorimeter is employed.

John C. Da Costa, of Philadelphia, described tanners' ulcers due to industrial chrome poisoning. T. M. Legge, of London, in 1902 collected many cases of perforated septums and chrome sores of the fingers. K. B. Lehmann, of Leipzig, in 1919 made studies of 722 persons engaged in the manufacture of chromates showing the toxic effects on the workers, as, perforation of the septum, ulcers of the skin and mucous membranes, as well as the pulminary and gastrointestinal organs. These same observations were made in 1901 by Hermanni, of Munich.

J. J. Bloomfield and W. Blum, U.S.P.H.S., in 1928 published HEALTH HAZARDS IN CHROMIUM PLATING, which also gives a good historical resume of chromium hazards.

Lead

THE best work on industrial plumbism was done in France in the early part of the nineteenth century, culminating in the great treatise by Tanqueral des Planchez, whom Alice Hamilton calls "The Columbus of Lead Poisoning." This astute clinician had the opportunity to observe 1217 cases at the Hospital de la Charite in Paris between 1831 and 1839. His contributions in the pathology of encephalopathy and contracted kidney are outstanding.

Another French investigator, Devergie, in 1838 observed the presence of lead in normal tissues. This initiated a controversy which continued unabated for nearly a century. Henry Burton, a Fellow of the Royal College of Surgeons at St. Thomas Hospital, in 1840 described his observations of the effect of the absorption by the gums. The blue line along the gums is an early sign of lead absorption. The discovery in very recent years of lead in the long bones by the use of the x-ray should be mentioned here.

J. J. Putnam, of Boston, in 1883 offered a method of diagnosing lead poisoning by detecting lead in the urine. In 1902 Sir Thomas Oliver, of Durham University in England, published his book DANGEROUS TRADES, contributing much to the knowledge of industrial diseases, especially in the field of lead poisoning, dusty trades, and accidents from live wires. J. C. Breton published, in 1911, his works on lead poisoning due to white lead, and on other occupational diseases. Sir Thomas Legge with K. W. Goadby—two outstanding English authorities on industrial hygiene—contributed in 1912 to the literature on lead poisoning and lead absorption. R. Craik in 1920 published the results of his observations on the effect of lead in erythrocytes. The basophile or stippled cells in blood examinations of lead workers offered an early diagnostic point in lead poisoning.

Anne S. Minot, of Boston, discovered that the greater part of lead in chronic plumbism is deposited and stored in the skeleton. This classic piece of research laid the foundation for the modern treatment of lead poisoning, and gave Harvard University, through the researches of J. C. Aub and his colleagues in 1924, the outstanding honors in lead studies. Later extensive work on lead in the human body was made by R. E. Kehoe, of the University of Cincinnati.

Outstanding contributions on this subject were the extensive reports in 1929 by the Committee on Lead Poisoning of the Section on Industrial Hygiene of the American Public Health Association. These reports appeared in a second mimeograph in 1930, and in subsequent issues of the Year Book. The basophilic aggregation test was developed in 1924 by Carey P. McCord, D. K. Minster, and M. Rehm, at the University of Cincinnati and the Industrial Health Conservancy Laboratories.

Mercury

MERCURY as an element was known by the ancient Egyptians in 1500 B. C. During the Roman period, Pliny and Dioscorides were acquainted with it, and later on the medieval alchemists named the planet for it. Paracelsus was the first to introduce mercury into medicine. Today this important element is widely used in the arts in making thermometers and mirrors, and in gilding. It is used in making pigments, antiseptics; and furriers and makers of felt hats find it necessary in their trades; it is used as amalgam in gold mining, and also is used in making fulminates.

The history of mercury in occupational diseases is very interesting. Fallopius noted that workers in quicksilver mines can endure the employment for barely three years. Andrew Mattioli, of Sienna, observed chronic mercurialism among the miners of Idria in the early half of the sixteenth century. Bernard de Jussein, in a memoir read to the Academy of Sciences in 1719, reported that the free workers in the mercurial mines at Almaden, since they were not restricted to the place and

were cleanly in their habits, had no disease other than a slight trembling, while the slaves, confined to dirty quarters, had swelling of the parotids, with aphthae, salivation pustules, scurvy and considerable trembling. Much later, in 1761, Giovanni Scopoli, of Alto Isonzo, wrote a clear description of mercurial tremor among miners of the same region. Ramazzini in his classic work on occupational diseases describes a case report in about the year 1700 of a young gilder who died without fever. He describes how his face became wan, his eyes swollen, the fetid stinking ulcers in his mouth, and the voiding of great quantities of ugly matter (salivation).

Etmuller stated that a quicksilver miner in four months becomes subject to tremors, palsy, and vertigo.

According to T. M. Legge, of London, mercurialism among hatters and furriers was first described by Reitz, of St. Petersburg, in 1829. A. Kussmaul, of Wurzburg, as early as 1861 noticed the manifestations of the nervous system in the form of tremors and changes in muscular coordination from mercurialism. He described a coppery red color of the throat, palate, and pharyngeal rings as a sign of chronic mercurial stomatitis. Kunkel, of Jena, in 1901 observed the toxicity of mercury and certain symptoms such as bloody stools, sometimes containing pieces of detached mucous membrane. G. Giglioli in 1909 noticed the presence of ptyalism as a marked feature in chronic mercurial poisoning. Both he and A. Kussmaul gave vivid descriptions of the nervous tremors and the various psychoses present. Tozzius remarked that miners of quicksilver lost their teeth and were subject to asthma. Samuel Berger, of Cleveland, Ohio, in 1932 advised flushing the bowel with water through a cecostomy operation in mercury poisoning.

In 1911 G. F. Gothlin, of Upsala University, determined that the minimum amount of mercury necessary to obviate mercurialism was 0.4 to 1.0 m. g. taken daily over a period of months. Ludwig Teleky, of Vienna, pointed out the relative receptiveness to mercury, especially among women workers, tubercular and anaemic individuals, in 1912. Estimating the amount of mercury from dust blown over in a hat factory to be about 5.56 m. g., daily, Lang, of Wiechaden, found in 1912 that of this only 0.0576 m. g. is soluble in the gastric juices.

In 1917 two German industrial hygienists, F. Koelsch and Ilzhofer, asserted that from 0.4 to 1 milligram of mercury taken daily for a month will produce mercury poisoning. Lloyd and Gradner, of London, in 1922 recognized the danger of exposure to small quantities of mercury. They estimated that .09 to .17% was contained in carrotted rabbits' fur.

Ravenant, a Frenchman, in 1920 had first called attention to sodium thiosulphate as an effective therapeutic agent in metallic poisoning. McBride and Dennie inroduced this into the United States in 1923. Alice Hamilton, of Boston, investigated the industrial diseases of fur cutters and hatters in 1922, and found that carrotters suffer much from gingivitis, tremors, and blackening and erosion of the teeth. This is from nitrate of mercury poisoning. She also investigated the workers at the quicksilver mines at New Almaden, California.

An effective antidote for acute mercury poisoning was developed by S. M. Rosenthal, U.S.P.H.S., 1933, called sodium formaldehyde sulphoxylate, which reduces mercuric corrosive chloride to an insoluble salt when taken internally.

Manganese

R. von JAKSCH, of Prague, described in 1901 the toxic symptoms of manganese: the emotional and neurological signs, the gait, and salivation symptoms. Another German, H. Embden, in the same year described his neurological observations, especially the multiple sclerosis and psychic symptoms. Before this time, Cooper, in 1837, had reported five cases of nervous diseases attributed to manganese poisoning.

L. Casamajor, of New York, in 1913 observed and studied 10 cases of an unusual form of mineral poisoning, attributing it to manganese, which affected the nervous system and locomotion of workers.

Dr. D. L. Edsall, of Harvard, noted that influenza mortality rates increased among workers in manganese, and in 1919, in collaboration with F. P. Wilbur and C. K. Drinker, of Boston, showed the occurrence, cause and prevention of chronic manganese poisoning.

Phosphorus

THE white or yellow phosphorus was discovered by Brandt, of Hamburg, in 1669, and the red or amorphous by Schroter, of Vienna, in 1845. John Walker, a druggist of Stockton in Tees, invented the lucifer match in 1827. Charles Sauria, a student at the College of Arc at Dole, in 1831 invented matches made from a mixture of phosphorus, sulphur, and chlorate of potash. Then Dr. Brocoorens, of Grammont, Belgium, reported a condition of fragility of bone among match workers.

Lucifer matches were first manufactured in 1833 from white phosphorus. Twelve years later Lorinser reported the phossy jaw, a new occupational disease in the form of a necrosis of the bone due to phosphorus. Dr. Lorinser diagnosed the first case of phosphorus necrosis in a match worker in 1838. In 1847 a study on diseases of match-makers, particularly the phosphorus necrosis, was published by von Belra and Geist, two German doctors. This frightful disease was eventually outlawed by legislation which substituted the sesquisulphide process for the use of white phosphorus. Sevene and Cahen, of France, developed the sesquisulphide of phosphorus match, which did not cause poisonous symptoms.

Dr. Derden, of Manchester, in 1899 reported a case where two match-dippers had fractured both thighs in a ridiculously simple way. He offered the opinion that these operators had a excess of phosphoric acid, which combined with the preexisting neutral phosphates of lime to form a slightly acid salt and thereby cause fragilites ossum.

Sir Thomas Oliver and Sir Thomas Legge were early English investigators who made inquiries into the dangers of using yellow and white phosphorus, during the early part of the twentieth century. J. B. Andrews, of Washington, in 1910 pointed out how fumes produce phossy jaw. Through his efforts, under the auspices of the American Association for Labor Legislation, a survey was made of the dreadful hazards of phosphorus poisoning in the match industry. His report interested President Taft, who requested the Diamond

Match Company to relinquish their patents on the sesquisulphide process. Congress awarded the company a vote of thanks for their generous action in doing so. A Federal law was also enacted in 1912 to abolish the phosphorus match.

Contributions to the literature on phosphorus poisoning in industry have been made by Teleky, of Vienna, Thomas Oliver, of England, J. B. Andrews and Alice Hamilton, of the United States.

Dr. Vallardi, of the Milan Clinic, could not produce necrosis of the jaw, the pathognomonic manifestation seen in humans, on animals with which he experimented.

Radium

IN 1898 Madame Marie Curie and her husband, Pierre, had isolated in Paris from pitch blende ore a radioactive element which she named radium. Both were awarded the Nobel Prize. Madame Curie, who was born in Warsaw and spent her entire life working with radium, died in 1934.

M. O. Wyes, a German, in 1906 was one of the early reporters of x-ray carcinoma among operators. H. S. Martland, of New Jersey, called attention in 1925 to the unrecognized dangers of handling radioactive substances; and in 1906 to certain anaemias due to radioactivity; and in 1929 that osteogenic sarcoma occurred in painters of watch dials using luminous paint.

In 1929 F. B. Flinn and S. M. Seidlin, of John Hopkins, inferred the use of parathormone in the treatment to eliminate radium from the body.

R. D. Evans, of the University of California, at Berkeley, reviewed the present knowledge in radium poisoning in 1933.

Sulphur

SULPHUR was known as one of the earliest elements. It is used in the manufacture of sulphuric acid, for bleaching and fumigating, and in insecticides. Regarding its irritation, Professor K. B. Lehmann and Ogata found that with sulphurous dioxide fumes it begins at the low point of 0.01 parts in 1000 parts of air. Susceptible persons are rendered ill by 0.03 parts, but tolerance is quickly established.

M. Patissier describes a disease known as "Anemie," which attacked epidemically for 11 years all the workmen in one gallery of a coal mine at Auzain, near Valenciennes. This gallery did not differ in situation or ventilation from others in its neighborhood. The air analyzed contained sulphuretted hydrogen and carbonic acid. The "Anemie" presented the following characters: violent colic, pain in the bowels and stomach, difficulty of breathing, palpitation, a prostration of strength, flatulence, and black and green colour of the faeces. This stage continued 10 or 12 hours and was succeeded by abdominal pains, weak pulse, concentrated and quickened, skin discoloured and tinted with yellow, locomotion difficult, and fatigue extreme, palpitations frequent, face swollen, sweats constant. The second stage was of many months' duration and was chiefly marked by depression and emaciation. At last the first symptoms recurred; there were dire pains in the head, frequent fainting, intolerance of light and sound, flatulence, purulent stools, sudden death. Less acute and urgent were the symptoms in persons who had not the first stage in a marked degree: and great bilious disorder with its attendant affections of the stomach, heart, and head, formed the chief character, and gave the name of "maladie jaune." On postmortem inspection, the arteries and veins were found to contain only a liquid serum.

Parent Duchatechet, of Paris, in 1829 investigated and reported on the hydrogen sulphide hazard in Paris sewers. He found as high as 2.99% gas present. Claude Bernard, of Paris, made an interesting experiment in 1857 when he

injected hydrogen sulphide solution into venous blood. He proved that the gas was eliminated through the lungs by showing the blackening of lead acetate when exposed to the exhaled air. In 1863 a German investigator, Hoppe Seyler, was the first to study the chemical action of hydrogen sulphide on the blood. He found that the oxyhaemoglobin formed a compound sulphine themoglobin.

That 0.1% of the gas is fatal was determined by H. Eulenberg in 1865 in researches with animals on the toxity of hydrogen sulphide. Thomas Oliver and R. A. Bolan, of England, declared in 1903 that in hydrogen sulphide poisoning the haemoglobin in slowly converted into methaemoglobin. R. von Jaksch, of Vienna, in 1910 showed that the symptoms are confined to the respiratory tract, in the form of a strangling cough, bloody mucous, cyanosis, croupous inflammation of the bronchi, and tubular pneumonia.

R. R. Sayers, Mitchell and W. P. Yant, of the United States Bureau of Mines, in 1923 found that so low a concentration of hydrogen sulphide as 0.005% would cause toxic symptoms, and on continued exposure at 0.02% would, if covering a number of days, cause death. Sayers recognized two types of industrial poisoning from this gas, the

acute symptoms with asphyxiation, and the subacute, with conjunctites and destructive action of the lining of the lungs with oedema.

Brass

BEFORE Solomon's time, the making of brass was known to the Phoenicians; and Pliny in the first century A.D. described brass fumes. The first description of brass founders' ague in English was published by Thackrah in his famous work *Essay on the Effect of Arts, Trades, and Professions on Health and Longevity,* written in 1830. Here he describes the symptoms of ague or intermittent fever which periodically attacks the brass worker. It remained, however, for Blandet, the Frenchman, to ascertain in 1845 that the poisonous fumes were due to the zinc in the brass. D. H. Greenhow in 1862 also recognized zinc as the important factor. Philip Drinker, of Boston, and his associates in 1927 showed that zinc chill, better known as fume fever, is a condition not produced by zinc alone, but is due to other combining substances, such as cadmium. C. C. Sturgis, P. Drinker, and R. M. Thomsen and associates, Boston, 1927, investigated metal fume fever by experimental inhalation of zinc oxide and noted resistance acquired by the inhalation on two successive days.

But to return to the industrial history: E. Hogben, of Birmingham, in 1887 reported on brass workers' disease, while M. Murray of England published a work on chronic brass poisoning in 1900.

The effect of methylene blue on the oxygen exchange in erythrocytes was first reported by Warburg in 1910. J. Haldane in 1912 used the colormetric method to determine saturation of haemoglobin with carbon monoxide. Lehmann and Saito in 1910 had proved that brass chills were due to zinc alone. In the same year E. R. Hayhurst, in connection with a survey he made for the Illinois Commission of Occupational Diseases, discussed the hazards of brass and zinc workers. This early American report was published by the commission in 1911.

Zinc was first smelted in England in 1743 and in the United States in 1873.

Carbon Monoxide

IN HIS history of carbon monoxide published in 1920, Dr. Lewin, of Berlin, has traced references to the action of this gas back into antiquity, and has shown that its effects have had a close relation to the history of the civilization of mankind. In ancient literature we find quotations which indicate that this poison was used in suicide, torture, and accidental death. Erasistratus, in 300 B.C., in Alexandria, assumed that the dilution of the air caused by vapors from coals was dangerous to the breather. Plutarch reports that the suicide of Catullus was by gas from glowing coals. In 68 A.D. Seneca ended his life by charcoal vapor. At the time of Cicero, poisoning by smoke was a form of punishment. It is said that the Emperor Septimus Severus (193-211) and the Emperor Diocletian (284-305) condemned many martyrs to death by this method and used the greenest and most smoke-producing wood possible for this purpose. Caelus Aurelianus described disturbances of movements and sensation due to carbon monoxide, in the middle of the third century. Erasistratus, in 300 B.C., had said that air diluted with vapors from coals destroyed the living tissue, and Avicenna, the Arab, in the tenth century called it a brain poison. Julian, the Apostate, 331-363 A.D., tells in one of his satires how he was nearly asphyxiated while in his winter quarters in Paris.

Herxheimer, a German investigator, was first to describe chlorine acne, doing so in 1899. M. C. Winternitz, then of the Chemical Warfare Service, described in 1920 experiments on dogs, showing the pathology of the respiratory tract as a result of chlorine gassing.

Carbon Dioxide

IT IS reported that carbon dioxide gas was discovered by J. B. Von Helmont, a Belgian physician who lived from 1577 to 1644. Industrially carbon dioxide gas is a by-product given off in fermentation, carbonated waters, and chemical fire extinguishers. Naturally it is found in caves and mines. It becomes a hazard to such workers as brewers and vinters; it is common in fermentation vats, silos, and linseed reservoirs. Black damp carbon dioxide, which is usually found in coal mines, consists, according to J. S. Haldane in 1902, of a mixture of 13% carbon dioxide and 87% nitrogen. When the oxygen is less than 12%, there are symptoms such as headache, deep breathing, clouding of the senses, and loss of consciousness. A lighted candle will not burn in such an atmosphere.

Cyanogen

THE cyanide compounds are used extensively in many trades such as electroplating, gold extraction, photo-engraving, manufacture of fumigants and fertilizers.

R. Kobert, of Stuttgart, in 1902 claimed that 0.3 parts of hydrocyanic acid in 1800 parts of air are fatal to man. R. von Jaksch, of Vienna, summarized in 1910 the toxic effects of hydrocyanic acid, its symptoms and pathology, saying that it causes internal suffocation in the presence of oxygen. Adding to the knowledge of pathology in cyanide poisoning, G. R. S. Thomas, of London, in 1923 published the autopsy findings in two fatal cases.

K. K. Chen, C. L. Rose, G. H. A. Clowes, 1933, of Eli Lilly and Company, Indianapolis, discovered the use of amyl nitrate as an antidote for cyanide poisoning, and in the same year W. B. Wendel, of the United States, used methylene blue in cyanide poisoning.

Benzol

MICHAEL FARADAY discovered benzol, or benzene, in illuminating gas in 1825. In 1842 Leigh found it in tar. Today benzol is the most important solvent in the arts; it is a by-product in making coke and is used for fuel. It is used at the starting point in the manufacture of drugs, dyes, etc.

An early autopsy account of a death by this poison was given in 1896 by F. Beinhauer. C. G. Stanesson, a Swedish toxicologist, at the International Congress at Moscow in 1897 brought to the attention of the physicians of the medical profession the toxic effects of benzol by reporting nine cases among girls using benzol rubber cement. Four of these cases ended in death.

In 1907 Wener Schultz, a German, described a clinical entity, leucopenia in agranulocytosis, that was destined to create confusion because of its marked similarity to benzol poisoning. L. Selling said in 1910 that the action of benzol on the bone marrow caused an anaemia, plastic, and a remarkable diminution of white blood cells. G. Y. Rusk,

of the University of California, in 1914, and L. Hektoen, of Chicago, in 1916, showed that its action on tissues and cells injured the production of antibodies and lessened resistance to bacterial infections. The hazards from spray painting, enameling, lacquering, etc., first recognized in 1915 by Dr. H. P. Albaugh, of Ohio, have enormously extended as innumerable poisons have come to be applied by this method.

Alice Hamilton brought the growing menace of benzol poisoning in American industry to the attention of the medical profession. She reported, in 1917, toxic jaundice from benzol and its derivatives. She found the condition prevalent among American munition workers. T. M. Legge, of London, also reported during this year on the high incidence of toxic jaundice and its mortality rate among T.N.T. workers.

T. E. Harington, of Boston, wrote on the toxic effects of benzene in 1917, and in the same year H. G. Weiskotten produced leucopenia in animals by exposing them to benzene vapor.

Irving Trifonow, a German, in 1922 developed a promising colormetric method for determination of benzol by the sensitivity of the per nitric reaction. The Shepard Gas Analysis Apparatus to investigate high benzol concentrations is used in conjunction with a freezing chamber of special design to prevent streamline flow of gas.

Paul Friedlander and C. P. McCord in 1926 noted experimentally an increase in basophilic material in blood of animals poisoned by benzol. McCord, of the Industrial Health Conservency Laboratory of Cincinnati, Ohio, published in 1931 a complete resume on benzol poisoning of a new investigation of the toxicity of benzene and benzene impurities —an epoch-making contribution.

While severe cases of benzol poisoning are not so numerous in the United States as formerly, the mortality rate is still high, and recovery is usually very slow. The usual treatment has been blood transfusion. However, in the *Journal of The American Medical Association* of December 21, 1929, a case of chronic benzol poisoning, treated with daily portions of calves' liver, after three transfusions were without benfit. This was reported by Adelaide Ross Smith, of New York.

Carbon Bisulphide

CARBON bisulphide was discovered by Sampadius, a Frieberg chemist, in 1796. It is used in the vulcanization of rubber, making paints, in artificial silk, and in rubber cements.

Payen, a Frenchman, in 1851 called attention to the danger of the poison. His countryman, Delpech, described in 1856 and again in 1863 the manifestations of excessive exaltation of the nervous system, with a general breakdown and moral and physical degeneration. He also gave the first comprehensive picture of its symptomatology. Tavera, of Paris, in 1865 studied the blood changes.

K. B. Lehmann, of Munich, in 1897 showed that one part of carbon bisulphide in 1000 parts of air will produce symptoms.

Marcilius Ficinus, 1433-1499, believed that all coals, especially extinguished coals, caused headache. Mercurialis in the sixteenth century believed that bad coal was the source of the trouble. Cassii, a Swiss, was the first to emphasize headache as a characteristic symptom, which he did in 1562; as a sign, however, it had been known to Aristotle.

Ambrose Pare, the great French barber surgeon, in 1575 described carbon monoxide poisoning. Reference was made by Marcelli Donate, of Venice, in 1588 to the peculiar coloring produced in the skin. Sir Francis Bacon in 1648 referred to this gas as vapor carbonium instead of fumes. J. B. Van Helmont is said to have named it carbon gas. H. Boerhaave, of Holland, probably made the first animal experiments with carbon monoxide in 1732. Cruickshank in 1800 showed that it was gaseous oxide of carbon as to symptoms and signs.

Samuel Witter, of Dublin, introduced in 1814 the use of oxygen inhalation in treatment for carbon monoxide poisoning. E. Klebe, the German, noted in 1865 the relaxation of muscles, dilatation of the pupil, and the peculiar anaemia due to this gas. First to prove that carbon monoxide displaces the oxygen in oxyhaemoglobin and causes the resulting cherry color, was Claude Bernard, the French physiologist, who made this discovery in

1857. L. Lewin, of Berlin, claims Troja in 1778 was first to call attention to the blood coloring. Moreau in 1869 brought the signs of so-called chronic carbon monoxide poisoning to the attention of the lay and medical public. He termed it the insanity of cooks, and ascribed it to working over poorly ventilated stoves.

Heiffner and Kulz were two German chemists who used the spectrophotometric method for detecting the gas for the first time in 1883. The Hoolamite, the activated iodine pentoxide indicator for testing air samples, was developed by C. R. Hoover in 1921. Y. Henderson and H. W. Haggard also in 1921 devised a 5% mixture of carbon dioxide with oxygen to eliminate the carbon monoxide

from the blood. These same investigators developed the H. H. Inhalator, an apparatus for administration of this gas mixture. Henderson estimated the concentrations of carbon monoxide necessary at a given time to produce symptoms. His researches were confirmed the following year by Sayers, Meriweather, and Yant, of the United States Bureau of Mines.

J. F. Heymans observed and demonstrated in 1922 that intravenous injections raise the temperature and increase the carbon dioxide output. He suggested that the effect of methylene blue was a catalyzer upon the oxidative processes. J. S. Haldane in 1922 showed that there is a general similarity between carbon monoxide and anoxemia. Sayers and Yant in 1923 developed the pyrotannic acid method to determine carbon monoxide in the blood and the air. M. C. Teague, of the same Bureau, devised the iodine pentoxide method for testing low concentrations in the air.

United States Public Health Bulletin 150, published in 1925, covers the literature on carbon monoxide to that date. A research worker at the University of California, M. M. Brooks, found in 1928 that animals unconscious from carbon monoxide regained consciousness in half the time when methylene blue solution in the proportion of 1% was intravenously injected. In 1932 J. C. Geiger, of San Francisco, first used methylene blue intravenously with success on a human victim of carbon monoxide poisoning.

Industrial Gases and Fumes

THE most important of the gases that have had historical interest as hazards to workers in mines, explosive works, and chemical industries will be briefly summarized under their respective headings. Such toxic gases as phosgene, used in chemical warfare, and hydrofluoric acid, used to etch glass, have been omitted.

It may be said that K. B. Lehmann, of Wurzburg, began in 1884 to make the most extensive quantitative studies of the most important gases, based on animal experimentation, observation in industry, and, when possible, with human subjects. His work has continued practically up to date.

Methane Gas

MARSH gas (CH_4) long known to miners as fire damp, is dangerous in mines because of its explosive qualities when mixed with oxygen. Whenever the quantity of more than two-tenths percent is present in the air, it may be detected by the cap of luminous flame in the safety lamp, known as the Burrel Test.

Haldane, of London, found in 1902 that since marsh gas is light, it diminishes the oxygen in the mine atmosphere, causing the miners to faint from lack of oxygen.

Nitrous Fumes

THESE poisonous fumes are given off in manufacturing nitric acid, in the nitration processes in making explosive, in the burning of films, and so forth. Haldane found that exposure to 0.5 parts of nitrous oxide in 1000 parts of air was fatal to mice. In experimenting with dogs, Hudson, of the E. I. Du Pont de Nemours Company, of Wilmington, Delaware, discovered that the fumes act locally as a caustic, causing congestion and oedema of the lungs.

Ammonia

AMMONIA is used extensively in refrigeration plants, ice factories, fertilizer works, and dye works.

The symptoms and pathology of severe poisoning from ammonia fumes were described by H. C. Fairbrother, of St. Louis, Missouri, in 1887. Dyspnea, continual coughing, oedema, and bronchial congestion were named as the symptoms. In 1909 E. Ronzani, a German, found that after prolonged inhalation of 0.5 in 1000 parts of air, there may be a loss of agglutinins and bactericidal substances in the blood, causing a loss of resistance; and there may be nutritional disturbances and irritation of the respiratory passages.

Chlorine

CHLORINE was discovered in the middle of the eighteenth century by K. W. Sheele, a Swedish apothecary. It is used in industry in the manufacture of bleaching powder, disinfecting of water, bleaching cloth and straw hats, and in gas warfare.

According to the researches of K. B. Lehmann in 1886, 0.01 parts per 1000 of air produces ulceration of the mucous membrane. K. B. Lehmann and E. Hoth believed that chloracne is due to excretion of chlorine through the skin. J. C. Wente, of Boston, in 1887 showed that chlorine gas acts as an irritant on the skin, producing prickling and redness, sometimes causing vesicles.

In 1899 the German, Landenheimer, published a study of carbon bisulphide poisoning among workers in the rubber industry. In 1902 F. C. Health, an American, described toxic amblyopis. Hauft, of Germany, in 1903 described the symptoms of poisoning in animals, the excitation followed by paralysis and death. Interstitial nephritis and pneumonia were produced. E. R. Hayhurst, of Ohio, in 1914 made investigations of the effects of carbon disulphide on rubber workers.

Ether

SULPHURIC ether came into prominence in the nineteenth century as the first inhaled anaesthetic for the relief of pain. Its use made surgery possible. Its merits were independently discovered by Dr. Crawford Long, of Georgia, who used it in 1842, and Dr. W. T. Morton, who introduced it into the Massachusetts General Hospital in 1846.

Ether presents an occupational hazard to munition workers in the manufacture of smokeless powder. During the Great War, Alice Hamilton, of the United States, and the Englishman, T. M. Legge, published their observations of its effects.

Petroleum

PETROLEUM as gasoline and naptha causes dizziness, confusion, nausea, and burning and smarting in the chest and eyes of workers who use it. Allen, of Great Britain, studied the effects of kerosene upon workers in the petrol industry in 1861. In 1901 the Frenchman, Dufour, described the "naptha jag", and in the same year Dorendorff, of Berlin, said that it caused numbness, pains, and loss of power in the limbs.

Wood Alcohol

WOOD alcohol, the toxicity of methyl alcohol and the ocular menace it produces were called to the attention of the medical profession by

J. M. Day, of Louisville, Kentucky, in 1896. C. A. Wood, of Chicago, and Buller, of Montreal, in 1904 made extensive studies of blindness and mortality caused by wood alcohol.

Explosives as an Industrial Hazard

G. E. LAWS described in the *Journal of the American Medical Association* in 1910 an industrial form of poisoning called "the nitroglycerine head."

Dr. G. E. Ebright, of San Francisco, after observing in 20 workers of nitroglycerine those susceptible to powder headache, concluded in 1914 that a form of immunity is established when workers are engaged continuously in the industry.

Rene Sands, of Belgium, in 1915 first reported the effect of ether on workers in the manufacture of smokeless powder.

Alice Hamilton, Special Investigator of Poisonous Industries, United States Bureau of Labor Statistics, in 1916, described the ether jags and various other symptoms found among workers. In 1917, while studying industrial poisons used in the manufacture of explosives, she found that the nitrous oxide fumes given off in the nitration process are the cause of many fatalities among workers.

Benjamin Moore, of London, a member of the British Medical Research, described in 1914 the causation and prevention of Trinitrotoluene poisoning.

B. H. Spilsburg, of London, in 1916 described the origin, symptoms, pathology and treatment of tetra-nitro-trinitrotoluene munition workers, and especially the prophylaxis of toxic jaundice.

A. Livingstone-Learmouth, and B. A. Cunningham, of London, gave a clinical account with the classifications of symptoms in Trinitrotoluene poisoning in 1916.

R. P. White, London, in that same year described the occupational dermatoses among tetra-nitro-trinitrotoluene operators; the staining, hardening and scaling of the skin.

In 1917 H. S. Hartland, of the United States, described the toxic jaundice and the acute yellow atrophy of the liver in tetra-nitro-trinitrotoluene fatalities.

S. R. Haythorn, of Pittsburgh, in 1918 conducted experimentally a masterpiece of pathological research in Trinitrotoluene poisoning.

G. R. Minot, of Boston, in 1919 emphasizes the importance of blood studies in cases of suspected tetra-nitro-trinitrotoluene poisoning, there being mild changes in the appearance of red cells and a decrease in the amount of haemoglobin.

T. M. Legge, His Majesty's Factory Inspector, London, 1920, describes the effect of nitrous oxide fumes, the resulting bronchitis and pneumonia, and the fatal termination.

Occupational Diseases Due To Biological Processes: Bacterial Infections, Hookworm, Cancer and Dermatology

IT WAS during the middle part of the nineteenth century that so much progress in the medical sciences was made, and these years marked the discovery of what is now the science of bacteriology. This science revolutionized medicine, and for the first time offered man a workable theory of the causes of diseases and the means by which they might be cured and prevented. It made possible modern surgery, public health, and food preservation. Although the Dutch microscopist, A. van Leeuwenhoek, of Delft, first saw protozoa in 1675 and bacteria in 1687, the Jesuit priest, A. Kircher, of Fulda, stated as early as 1658 the doctrine of a contagium animatum as the cause of infectious disease. M. A. von Plenciz, of Vienna, in 1762 advanced the idea that each disease had a special contagium animatum, and J. Henle, of Germany, in 1840 announced the theory of contagium animatum.

The founder of bacteriology was Louis Pasteur of France, who lived from 1822 to 1895. Being a chemist, his first interest was in the field of crystallography. This led him to the process of fermentation and the ultimate discovery of micro-organisms as a causative agent. His studies in the diseases of wines and beers, silk worms, anthrax, and chicken cholera, opened up the field of bacterial research and preventive vaccinations for anthrax and rabies. He discovered micro-organisms of surgical infections in 1878.

Robert Koch, of Berlin, 1843-1910, laid the foundation for the theory of the development of infectious diseases. He worked out the anthrax life history in 1876, and described the etiology of traumatic infectious diseases. He discovered tubercle baccillus in 1882 and the cholera vibrio the following year.

It was the work of these two men and the wonderful associates they attracted to their laboratories that virtually abolished several communicable diseases, developed modern sanitation, and increased man's span of life. The discovery of various bacteria and protozoa of special interest to the industrial physician and hygienist followed: typhoid was discovered by C. J. Eberth in 1880; malaria by A. Laverin in the same year; glanders by F. Loefler in 1882; erysipelas by F. Fehleisen in 1883; tetanus by A. Nicolaier in 1884; bacillus aerogenes infection by W. H. Welch and G. H. F. Nuttall in 1892; dysentery by K. Shiga in 1897.

The methods of transferring pathogenic organisms by means of insects and other vectars being discovered, it opened the way for modern hygiene. This was a great aid to the sanitarian in temporary industrial and military camps. Theobald Smith in 1889 blazed the way in the discovery of the transmission of disease by an intermediate host, giving a new direction to the investigation of certain communicable diseases. Under this stimulation the anophele and aedes mosquitoes were soon incriminated as the transmitters of malaria and yellow fever, through the efforts of Patrick Manson and Sir Ronald Ross, of Great Britain, and Carlos J. Finlay, of Cuba, and Walter Reed, of the United States. This made it possible for William C. Gorgas to promote mosquito control measures in the Panama Canal zone, and to eliminate the fearful scourges which had prevented the building of the canal by the French.

Occupational Diseases Due to Infective Organisms

IN CERTAIN occupations the workers become infected by handling diseased animals or their tissues. Such trades are those of butchers, slaughter house workers, farmers, herders, wool sorters, hair and brush makers, hide and leather workers.

Anthrax

ANTHRAX is one of the most common of these diseases. It was known to the ancients as carbuncle, and was the first occupational disease discovered to be due to bacteria. P. F. O. Rayer discovered anthrax among hair workers in 1835 and in 1837 glanders in man. Lawrence, of England, described the condition among similar workers in 1847. In 1849 Pollender, while studying the blood of animals dead from anthrax, discovered numerous rod-shaped microscopic bodies, which he claimed were the cause of the disease. C. Devaine showed that the blood of an animal when injected in another could not cause anthrax unless it contained these bodies. He gave them the name of Baccilli Anthracis. His views were not immediately accepted but were substantiated when Robert Koch in 1876 isolated the baccilli in pure culture and demonstrated their ability to form spores. L. Pasteur and R. Koch simultaneously and independently demonstrated beyond doubt that the germ of anthrax is actually the cause of this disease by cultivating it in pure cultures and artificially infecting animals by its use. In 1881 Louis Pasteur produced a vaccine against anthrax.

Spear and Bell, of the Bradford district in Yorkshire, in 1881 described wool sorters' disease. Sclavo, of Milan, in 1903 introduced anti-anthrax serum.

T. M. Legge in 1905 gathered together nearly all the facts known about wool sorters' disease. In 1919 he declared that in the previous four years the mortality rate among wool, hair, hide and skin workers was 15%.

J. B. Andrews, secretary of the American Federation of Labor, pioneered a movement in America for the prevention of anthrax infection.

Undulant Fever

UNDULANT fever was described by Hippocrates in his book on Epidemics. J. A. Marston in 1859 wrote an accurate and detailed account of the disease as it existed in Malta. D. Bruce in 1893 discovered the micrococcus and named it the micrococcus melitensis. M. L. Hughes in 1896 designated the disease as undulant fever. A. C. Evans in 1918 demonstrated the close relationship between undulant and infectious abortion in animals.

Tularemia

TULAREMIA has lately been identified as a disease; yet in Leviticus there is a passage, "And the coney . . . and the hare . . . of their flesh shall ye not eat and their carcass shall ye not touch, they are unclean to you." G. W. McCoy first described the diseases among rodents in Tulare County, California. The name of the disease was taken from the word tules, which grow in the marshes in that county. In 1912 G. W. McCoy and C. W. Chapin, of the United States Public Health Service, discovered the organism Bacterium Tularense. The researches and contributions of Edward Francis in 1921 are outstanding in this field.

Rocky Mountain Fever

MAJOR W. W. WOOD, a United States army man, made the first report of the prevalence of Rocky Mountain spotted fever in 1896 among civilians about the Boise barracks. H. T. Ricketts, of Chicago, in 1907 demonstrated that infected tick bites produced the disease, and found a micro-organism which had a specific relationship to Rocky Mountain spotted fever, classed as Rickettsia. R. R. Spencer, U.S.P.H.S., produced vaccine in 1924, for the prevention of this disease.

Bone Button Infections

AN INDUSTRIAL disease of the skin known as "diamond skin disease" among workers in bone button industry is due to bacillus of swine erysipelas; this disease affects bone handlers, bone meal workers, fishermen and button manufacturers. It was recognized by Louis Pasteur in 1882 and confirmed and described by F. Loeffler in 1885. In 1891 Jensen described diamond-shaped skin lesions in hogs due to bacillus of swine erysipelas. W. W. Stefansky and A. A. Grunfeld in 1930 reported two hundred cases of infection with bacillus of swine erysipelas occurring among fresh water fish handlers in Odessa. G. B. Lawson and M. S. Stinnett in 1931 described symptoms during an outbreak in a button factory leading to a diagnosis of erysipeloid, probably due to bacillus of swine erysipelas. F. F. McGinnis, of Richmond, Va., advises sterilization of bones at a temperature of 144 degrees to destroy bacillus of swine erysipelas (erysepothux Rhusiopathiae).

Vaccinia

COWPOX (variola vaccinia) is a disease chiefly of milch cows and originated in all probability from human smallpox some time in the past centuries. The disease during Edward Jenner's time, 1780, was prevalent among dairy maids and cattlemen, just as smallpox was common in England at that period.

Spirochetosis

SPIROCHETAL jaundice is a common disease among Japanese miners. It carries a mortality of 40%. The organism is the Leptospera icterohemorrhagiae which was discovered in infected rodents and humans' blood in 1914 by two Japanese investigators, R. Triada and Y. Ido. The spirochetes have been found in the urine of mine rats.

This disease was prevalent among soldiers in the trenches during the great war.

Pier D. Siccardi, an associate of Professor Devoto of the Milan Clinic, scientifically studied icterhemorrhagic spirochetosis, which he discovered among troops in the Fourth Italian Army. He succumbed to this disease during the Great War.

Hookworm

LUCRETIUS and Lucan, the one living around the year 50 B.C. and the other around the year 50 A.D., frequently mention in their classical works the striking pallor of miners; this probably was due to hookworm infection.

Froelich in 1789 found the so-called hooks and gave the name hookworm or uncinaria to the disease. James Thomson described in 1820 the havoc it wrought among slave workers in Jamaica, due to Necator Americanus. Angelo Dubini, an Italian physician living at Milan, in 1838, discovered what he named the ankylostoma duodenale in a human body. Then Buzzolo, of Turin, in 1879 successfully tried thymol as a vermifuge among brickmakers.

Perroncito, of the University of Turin, found in 1880 that the epidemic of anaemia among the workers in the St. Gotthard Tunnel was due to

hookworm. Miners' anaemia is due to hookworm disease and was first found among the Cornish miners who brought it to the mother lode in California. E. Bugnion in 1881 was also attracted by its prevalence among the workers on the St. Gotthard Tunnel through the Alps. J. Haldane observed an outbreak in a Cornish mine in 1903. Arthur Loossin Cairo had accidentally discovered in 1898 that the skin was the channel of infection in hookworm disease. This revolutionized the methods of control. C. W. Stiles, of Washington, D. C., discovered the America specie, Uncinaria Americana, in 1902.

Notable public health activities in campaigning against hookworm were carried on at Porto Rico in 1899 by Colonel B. K. Ashford, U.S.A., and by the Rockefeller Sanitary Commission organized for hookworm prevention in 1910. Extension of hookworm control was carried out in many parts of the world by the International Health Board beginning in 1915. This same board published in 1922 a bibliography of hookworm diseases.

F. F. Sprague, of Jackson, California, recognized in 1906 the prevalence of hookworm in the mother lode in California. This was confirmed by Dr. Herbert Gunn, of San Francisco, three years later.

Actinomycosis and Glanders

THE RAY FUNGUS in man was discovered by von Langenbeck in 1848. O. Bollinger in 1876 found that lumpy jaw among cattle, which had been recognized since 1785, was due to a fungus. In 1878 J. Israel found the parasite in man. E. Ponfick in 1879 pointed out the identity of the human and bovine infection. Schilling in 1821 demonstrated the transmission of glanders to man. P. F. O. Rayer in 1837 established glanders as a specific disease in man. F. Loeffler of Prussia in 1882 discovered the bacillus of glanders.

Industrial Tuberculosis

TUBERCULOSIS Verrucosa Cutis, a common warty condition of the skin due to the handling of tuberculous meat and bodies, and an occupational disease found among pathologists and meat handlers, was first described by Samuel Wilkes in 1862.

Dr. Greenhow, in an analysis of the statistics of lung diseases in agriculture and manufacturing populations, reported during 1859-1862 to the English Board of Health and the Privy Council, that when the workers are attracted indoors the mortality of phthisis is increased. J. A. Villemin in 1865 demonstrated the specificity of tuberculosis and in summary said that tuberculosis is caused by an inoculable agent. Then Robert Koch in 1882 demonstrated to the world that he had discovered the tubercle bacillus.

Dr. Frank T. Fulton, of Providence, Rhode Island, as early as 1906 introduced physical examinations in a saw works in an effort to isolate tuberculous workers.

P. Reckseh, of Berlin, concluded as early as 1903 that statistics in lime-producing districts show tuberculosis to be rare, and when present its course is favorably affected. B. Silkirk, of Edinburgh, in 1908 was unable to find among lime and cement workers in his district any record of a worker having died of tuberculosis, and suggested a lime works as a curative for a tuberculous colony.

Three important statistical studies have been made in the United States on the ratio of tuberculosis deaths to the total deaths in various industries where dust is a problem. The first of these, conducted by the United States Bureau of the Census, gave the proportionate mortality in occupations of the population of the registration area for 1908-1909. Second was a study on the industrial experiences of the Metropolitan Life Insurance Company by Dr. Louis I. Dublin. The third and most extensive collection of mortality statistics in dusty trades was compiled by Dr. Frederick L. Hoffman of the Prudential Life Insurance Company in 1918.

T. B. Sachs, of the Chicago Tuberculosis Institute, pioneered a movement in 1911 which planned for the early detection of tuberculosis among workers in Chicago factories. In 1913 E. L. Collis, M.D., gained distinction by his studies of physiological and pathological effects in regard to the action of dust in producing lung disease, and in 1914 published his findings in regard to housing as a factor in tuberculosis causation. In 1921 he published in collaboration with Major Greenwood, "The Health of the Industrial Worker", giving a satisfactory account from the standpoint of industrial hygiene of the changes in civilized life in the past century as a result of the rising development of modern industry.

The Miners' Phthisis Prevention Committee of South Africa in 1919 showed that fine rock dust produced by drilling in quartz produced silicosis, a fibrosis of the lung, tuberculosis being superimposed. Contributions on tuberculosis in the dusty trades are given further consideration under the heading of pneumoconiosis.

One of the most active participants in the movement to combat tuberculosis in industry after public sentiment was aroused against this disease was a committee on factories of the Chicago Tuberculosis Institute, made up of members of the profession under the guidance of Dr. Sachs, mentioned above, who introduced physical examinations for the workers.

A recent study of Ohio vital statistics showed that, *in mechanical and manufacturing industries*, the tuberculosis death rate for females was 118.1 as compared to 96.6 for males, for the year 1929, and that, in 1930, among those so employed the ratio of female deaths to males was nearly six to one (*Ohio Health News*, VIII, 19:3, October 1, 1932).

The recent monograph, "Death Rates and Occupations", by Jessamine S. Whitney of the National Tuberculosis Association, shows that tuberculosis was the second cause of death among *gainfully occupied males* in 1930 with a rate of 95.1 per 1,000,000, or next to heart disease.

Industrial Cancer

IT IS NOW established that workers with certain elements have a larger incidence of morbidity due to cancer. How these agents influence a susceptibility to malignancy is not at the present understood.

Arsenical cancer was witnessed in 1825 in Paris among industrial workers in tin and copper, and in Leipzig in 1850 among parafin workers.

The German surgeon, Pelm, in 1907 called attention to twenty-one cases of tumor of the bladder, mostly of the papillomatous and malignant types, which he had collected among aniline workers. Reasearch into occupational cancer mortality

was carried out by Greenwood in 1919. In 1922, in Manchester, England, A. H. Southam and S. R. Wilson found a new type of scrotal cancer known as Mule spinners' cancer among workers in cotton mills. This was caused by oil made from shale, which carries a carcinogenic substance, that came in contact with the scrotum while the worker was operating the machine, due to soiling of trousers.

H. S. Martland, of New Jersey, first described in 1925 and 1929 the cases of splastic anaemia with blue necrosis in women who had been exposed to the action of alpha rays from swallowing luminous paint while employed in painting watch dials. Later he published the reports on the development of bone sarcoma in the severely poisoned.

H. Oppenheimer, of Germany, in 1926, reported cancer of the posterior urethra and prostate in workers in the chemical trades handling aniline—also cases of pailloma in the bladder.

P. A. Weber, of Saxony, 1927, analysed the Schneeberg miners whose cases were investigated by the Cancer Research Committee of Saxony and found the lung cancer was due to the dust of the mine which contains arsenic and radioactive elements.

C. C. Twort and J. M. Twort, of Manchester, England, in 1930, investigated the relative potency of carcinogenic tars and oils. Mule spinners' scrotal cancer is found among the workers in cotton mills where lubricating oils are used. In 1934 the Tworts claimed that the cancer producing property of the mineral oil is closely related to its refractivity constant. The English working man, investigators report, leads in skin cancer from the handling of briquets made of coal dust burned with gas works tar.

The English Registrar General shows by figures that there is some connection between alcoholism and excess of cancer, and that brewers and others engaged in the manufacture and sale of alcohol suffer from excess of cancer. Malignant disease of the bladder has been found to be 33 times as frequent in Basel among workers in fuchcin and aniline factories as in the general male population.

The report of the Departmental Committee on Compensation for Industrial Diseases, in 1907, recognized epitheliomatous ulceration due to pitch, tar and tarry compounds among patent fuel workers in South Wales.

Industrial Dermatology

OCCUPATIONAL dermatoses are recognized as by far the most widespread of characteristic occupational diseases, and the group of oils, greases and cutting compounds usually leads, with cleaning agents and petroleum distillates making a close second. The primary cause is due to mechanical obstruction of the sebaceous orifices, by the extraneous matter suspended in the oil, which becomes infected by skin organisms or through minute traumas of the skin. Remy and Zorea, of Paris, first reported this form of dermatosis, although the effects of irritants of other trades upon the skin had been described in 1806 by J. L. Alibert, and Willen, of England, was familiar in 1817 with "bakers' itch", which he named psoriasis diffusa. Thomas Bateman, of London, a pupil of Willen, in 1835 pursued dermatological studies on the effects of occupational diseases upon the skin.

Ibrelisle, of Vienna, studied skin diseases of handlers of human and animal hair (anthrax), and advocated sterilization of the hair. The Viennese School of Dermatology was founded in 1840 by Hebra, and his pupils Kaposi and Neumann engaged in fruitful researches there.

Thouvenin, of France, wrote in 1846 on skin diseases contracted in the woolen industry. The two French investigators Mehier and Chevallier studied the affections of the skin among mother of pearl workers in 1852. Chevallier was a distinguished pharmacist. He early called attention to the relative effects of certain chemicals in producing skin diseases, and studied the effect of bichromate of potassium.

Themlier wrote, in 1864, on skin affections of laboratory workers. Giraudeau, Gilbert, Saint-Gervais, and Devergie published monographs on skin diseases in this year, while Bazin treated in an article the skin diseases of many kinds of industrial workers. Dankwerth, the German, in 1868 wrote an article on petroleum eczema. Leloir, of Lille, in 1885 reported a dermatitis among flax spinners. In 1886 Harrington, of Boston, described dermatatis due to chrome. White, of Boston, in 1887 published his notes on Dermatitis Venenata. An article on cutaneous poisoning from petroleum was published by L. Lewin in 1888. Herxheimer in 1899 described for the first time chlorine acne. J. Schamberg in 1909 described grain itch and found it to be caused by a mite pediculoides ventricosus.

In 1915 R. Prosser White, of London, wrote the first edition of his book, OCCUPATIONAL DERMATOLOGY. The first dermatological clinic was instituted at the Broom Street Infirmary in New York. In 1918 F. E. Deeds, U. S. Department of Labor, at a Chicago steel plant investigated an epidemic of furunculosis among handlers of steel who cleaned their hands with paraffin oil. B. F. Davis described lesions due to paraffin oil among press workers in 1920. "Oil boils" or "Bouton D'Huile" were described by Kober and Hayhurst in 1924.

L. Schwartz, United States Public Health Service, 1932, reported that industrial dermatitis or occupational eczema contributes about 50% of all claims for compensation due to conditions classified as industrial diseases.

H. Goodman, of New York, concluded in 1933 that silk handlers' dermatitis is due to the anti-mildew solution which is similar to crude phenol.

Skull and Brain Injuries

By W. H. BLAKE, JR., M.D.,
Sheffield, Alabama

THIS is a common injury, seen frequently by men doing traumatic, industrial and general surgery.* Being one of the designated surgeons to do the work for the Government and the Tennessee Valley Authority, I want to congratulate the Staff of the TVA. for the small death rate and comparatively few serious injuries they have had thus far in their work around Muscle Shoals. I believe just as important work lies ahead in preventive accidental work as in preventive medicine.

This paper will not deal with birth injuries, although I am sure all of us can recall sad experiences with such cases.

* Presented at the Thirty-Ninth Annual Meeting of the Association of Surgeons of the Southern Railway System, Knoxville, Tenn., June 16-18, 1936.

Basal fractures, in our experience, have carried a death rate of 50%. Other skull fractures have been only 20%. Our series is made up of only 54 cases. Crandon and Wilson collected 540 basal fractures with 262 deaths, or practically the same percentage.

The principal symptoms and physical findings in skull fractures are hemorrhage, escape of cerebrospinal fluid or of brain matter, the exit of air from the sinuses into the surrounding tissues, cranial berve and pupillary disturbances, circulatory, respiratory and temperature changes. These, with the stereoscopic x-ray plates, make the diagnosis. The base fractures being so much more serious, for convenience of diagnosis and description, the base of the skull is divided into three fossae known as the anterior, middle and posterior. Hemorrhage from the nose and mouth generally indicates a fracture in the anterior fossa involving the cribriform plate of the ethmoid. Fractures of the middle fossa usually cause bleeding from the ears and mouth. The reason for this is that most of these fractures involve the external auditory canal or the inner and anterior wall of the middle ear. Profuse and long continued hemorrhage from the ear may mean injury to the lateral sinus or middle meningeal artery.

Crandon and Wilson found that in a large collection of cases the mortality was 66% when the hemorrhage was from the ears, 61% when it was from the nose, 39% when it was from one ear, and 33% when it was from the mouth. Therefore this conclusion seems logical: if the bleeding is from both ears or from the nose in base fractures, the chances of recovery are two to one against the patient; whereas, if the bleeding is from one ear or from the mouth, his chances are two to one for recovery. This is, of course, only for prognostic purposes, or fairly accurate data to tell the people, as they will certainly want to know what you think the chances are for either life, death, or permanent disabilities.

Hemorrhage in the posterior fossa is usually late in developing and makes itself evident by a boggy ecchymotic area around the posterior surface of the scalp, neck and mastoid region.

If cerebrospinal fluid escapes from the ears, nose or mouth, this indicates a fracture with rupture of the dura and arachnoid membrane, as the source of the fluid is, of course, from the subarachnoid spaces. This happens in about 5% of the cases This escape may be profuse. Stimson reports a case in which 63 ounces escaped in four and one-half days. Sir Wm. MacCormac reports a case that seems incredible in which he claims 10 pints were eliminated within five hours. The escape of brain tissue, except in crushing or shot wounds, is rare—one case reported in 300.

If one of the air sinuses is fractured there may be diffuse or circumscribed emphysema, which may be made worse by sneezing or forced breathing The most common sinus fractured is the frontal.

Cranial nerve involvements are common, and frequently due to the fracture extending to the canal through which the nerve makes its exit from the skull; or to pressure from bone, bloodclots, etc. The nerve involvement is apt to be immediate. A secondary paralysis frequently means infection and, of course, is a grave omen. If the nerve is cut in the region of the ganglion toe paralysis is permanent If due to pressure on or hemorrhage into the nerve sheath the condition clears up, with absorption or removal of the cause of the pressure if done reasonably soon. The facial nerve is the most common one involved.

Regarding the pupillary changes, they may be either dilated, contracted or equal. They were contracted on the affected side in 36% of the cases and dilated in 18%. They were equal in 46%. So we think very little can be deduced from the pupils.

Temperature changes in these patients are a very striking finding, and of inestimable value from a prognostic standpoint, if understood. One finding is that, at first, the temperature is always subnormal. Statistics show that as regards the prognosis all skull fractures may be divided into four groups according to their temperature chart:

1. If the temperature is first subnormal and undergoes a rapid and progressive rise, the prognosis is unfavorable.

2. If the temperature stays subnormal and you are unable, after several hours, by stimulation, hot packs, etc., to elevate it, the prognosis is unfavorable.

3. The prognosis is favorable if, after the shock, the previously subnormal temperature rises to normal and remains so.

4. If the temperature is first subnormal, rises gradually to 101° or 102°, marks time for several days and then declines to normal, the prognosis is good; but if after remaining around 102° it later begins to rise, there is evidently an infection and the prognosis is unfavorable.

Probably the most important thing that a surgeon should make himself certain of is the diagnosis and treatment of intracranial hemorrhage. This might take place from any of the intracranial vessels or sinuses, but by far the most common is from the middle meningeal artery. No guesswork or expectant treatment should be allowed, for it is here that a surgeon can be a life-saver and has an opportunity for real scientific service that is seldom offered elsewhere. Cushing considers the mortality 90% in cases of meningeal hemorrhage untreated surgically and 23% if the vessel is properly ligated and drainage instituted. Hemorrhage from the middle meningeal artery, either from its anterior or posterior branch, occurs in 27% of skull fractures and 45% of those of the middle fossa of the base.

The symptoms and findings of intracranial hemorrhage are: A lucid interval of consciousness following the primary unconsciousness before the unconsciousness of compression occurs. We have had several cases of this type we were able to save, and two I feel sure would have lived had the clot been removed sooner. At operation large organized clots were easily removed, but the pressure had existed several days with fatal brain damage before we saw them. In others seen shortly after the injury much larger clots and portions of bone were removed with very gratifying results. If the symptoms above are not recognized early then gradual pulse changes take place. At first it is slow with a slight or marked increase in blood pressure. This is due to pressure on the vagus center in the medulla. If this hemorrhage is allowed to continue the vagus becomes exhausted and then the pulse becomes very rapid, with a decided drop in blood pressure. We should not allow our patients to go that far, and certainly if the pressure starts dropping and the

pulse running, our chances of saving them are greatly diminished.

There are similar respiratory changes, but time does not permit me to discuss them.

The proper locations of the main vessels are, therefore, very important. The anterior meningeal is in the temporal region, one inch superior and anterior to the external auditory canal. The posterior branch is one inch superior and posterior to the same canal.

If the skull is crushed in, of course a decompression is always indicated. It is frequently wise to wait until the shock of the injury has been overcome. The risk of infection through ears, nose, and mouth may be lessened by antiseptic swabs; no syringes or packs should be used. Urotropin shows up in the spinal fluid within 30 minutes after administration by mouth; hence it should be given in doses of 10 to 20 gr. per day, until all chances or signs of infection have disappeared.

In simple, uncomplicated, closed fractures the treatment is very simple. Place the patient in a quiet dark room with a constant attendant as they are frequently delirious; with orders to chart the pulse, respiration, and temperature each hour and to notify you of any marked change. It sometimes requires much self control to do what is best for this type, because the people are wanting something more drastic done. I am certain too many surgeons are doing much unnecessary and harmful operating; not only on the skull but more frequently on the abdomen and pelvis. So it requires, as does the streptococcic pelvis, heroic waiting and symptomatic treatments. Stimulation, sedatives, enemas, catheterization, and other symptomatic measures should be promptly employed. Lumbar puncture is advocated by some, but is too dangerous to use except in selected cases. Watch the patient closely and be ever ready to do a decompression or ligation, night or day, if symptoms of compression, known by the given classical signs, make themselves evident.

The Accident Rate

—Its Relation to the Day of the Week—

By R. D. MUDD, M.D.,
Plant Physician, Chevrolet and Fisher Body, Norwood, Ohio

THE relation of the day of the week to the accident rate must be a source of speculation to every industrial physician. The answer has some practical importnace in that, if a certain day can be expected to produce more accidents than another, more caution should be exercised on that day, and more medical supervision should be in readiness.

Of course, it is admitted that statistics on this question must of necessity be subject to considerable variation, and no one isolated experience can furnish the final answer. The results of a study of this problem are published in the hope that it will stimulate a similar study among other industrial physicians.

The period of time covered by this study extended from July 29, 1935, to December 23, 1935, excluding the period August 27 to September 25 (during which time the plant in question was not in operation). The period selected was used because each day was remarkably constant for the number of hours worked and the number of employees on the job. This is shown by the following:

16 Mondays the average of man hours was	7614
16 Tuesdays the average of man hours was	7447
15 Wednesdays the average of man hours was	7552
15 Thursdays the average of man hours was	7902
16 Fridays the average of man hours was	7602

(Note: One Wednesday was omitted because it was a half day and one Thursday because it was a holiday.)

Every injury no matter how small was counted. This would appear to be the only satisfactory procedure, for otherwise there would be a disagreement as to what accidents should be counted.

The total number of such accidents occuring was as follows:

16 Mondays	463
16 Tuesdays	440
15 Wednesdays	403
15 Thursdays	443
16 Fridays	394

Reducing these accidents to the number per 1000 nine-hour workers, gives the following:

Day of Week	No. of Accidents Per 1000 nine-hour workers
Monday	34.15
Tuesday	33.20
Wednesday	32.17
Thursday	32.84
Friday	29.04

The study shows very clearly that the last day of the working period was productive of the smallest number of accidents. This is not due to failure to report minor accidents (because in my experience, the employee is more careful than usual to report Friday's accidents, as he does not want anything to happen which might interfere with the enjoyment of the week end period; and he does not want to return on Saturday to make a report of any accident.)

My interpretation of the results of this study is as follows:

MONDAY: Is the most dangerous day because the men have lost their stride. They are, in a small way, learning their jobs anew. In addition, a fair number of employees lose more sleep than usual over the week end, and more than the usual glass of beer is imbibed.

TUESDAY: The conditions of Monday are present to a less extent. The employee obtained a good night's rest and is on guard to a greater extent.

WEDNESDAY: He has attained his "full stride"—and has now had two full nights' sleep.

THURSDAY: The average employee may be a little tired. His work may be somewhat monotonous to him and he is less careful. As a result, the accident rate climbs a little.

FRIDAY: Being pay day, and with a week end holiday in mind, the employee is pepped up; he is more alert, more careful and the accident rate falls.

This study did not take into consideration the question of severity. Severity would probably have to be determined, to a great extent at least, by loss of time. With adequate full time industrial medical care, the number of lost time accidents can hardly be a guide as to severity. Until standards for determining the severity of injuries which do not result in loss of time, are available, such a study will probably have but questionable value.

Dust Hazards in Industry

THE campaign for prevention and ultimate elimination of silicosis and other dust hazards in New York State industries as provided by legislation of the last session may be said to have gotten under way this month with the appointment by State Industrial Commissioner Elmer F. Andrews of an Advisory Committee to make a comprehensive study and recommendations upon dust-collecting and eradicating devices, and rules and regulations for the prevention of dust diseases hazards in rock drilling, and to report thereupon to the Industrial Board.

Appointed to this Committee are:

DR. LEROY U. GARDNER, Director, The Saranac Laboratory for Study of Tuberculosis, Saranac Lake, N. Y.; Alternate, Dr. Sampson, Assistant Medical Director, same.

DR. A. J. LANZA, Assistant Medical Director, Metropolitan Life Insurance Co., 1 Madison Ave., New York City; Alternate, Dr. W. J. McConnell, Director, Industrial Health Section.

DR. WENDELL J. STAINSBY, Assistant Professor of Medicine, Cornell University Medical School, 1300 York Ave., New York City.

There will also be representatives of the Rochester Medical School, Rochester, N. Y., and The Office of the Surgeon General, U. S. Public Health Service, Washington, D. C.

The first meeting of this Advisory Committee took place at 80 Centre Street, New York City, on Thursday, July 30, when there was a general discussion of the problems involved and a plan of procedure for the Committee's work was outlined. It will first be necessary for the Committee to arrive at a definition of what constitutes harmful dust and what constitutes a harmful air content of harmful dust. Formulation of these definitions was assigned to a sub-committee consisting of physicians. Existing Industrial Code rules and regulations will be taken as a basis for rules and regulations aimed at the dust disease hazards, where possible the rules being modified and extended to cover the dust hazard requirements.

When the Advisory Committee has made its findings it will report to the Industrial Board which will give them further consideration and determine whether or not they should be included in the Industrial Code. The dust hazard amendments to the Industrial Code will become effective 20 days after they are first published following approval of the Industrial Commissioner.

Appointment of the Advisory Committee followed shortly a warning sent to employers in the dust hazard industries of the State by Industrial Commissioner Andrews against final commitment, acceptance and installation of dust-collecting and eradicating machinery before such machinery had been formally approved by the Industrial Board.

Pain

By W. S. NASH, M.D.
Knoxville, Tennessee

FROM the very beginning of the scientific study of living tissues it was accurately determined that there were certain resident and inherent properties in all living tissue.* After careful study these properties were classified and were found to be almost universally the same.

For example there were found to be in a living cell the inherent properties of irritability, spontaneity, contractility and reproduction. It was not always possible to work out all these attributes in a single living cell, but it was possible to work out some of them in one cell and others in another cell. Probably had our investigation been minute enough and thorough enough, and had we had suitable instruments for correct and proper determination, we might have found all the attributes in a single cell.

In mono-cellular life all these attributes have been determined under conditions which favored the determination of the several properties attributed to a cell. Mono-cell life is very simple, and yet, withal, it shows unmistakable evidences of contractility, of irritability, spontaneity and reproduction.

The irritability and spontaneity of early cell life are the attributes to be discussed in this paper—more especially that attribute of simple cell life spoken of as irritability. These inherent properties in protoplasmic life may be described as the very beginning of the more intricate part of a living complex body. When influenced by an external stimulant no matter of what character the protoplasmic element changes its shape and proceeds to move in a direction which is compatible with its preservation and environment; through inherency it may do the same thing, and so we have hand in hand spontaneity and irritability well defined and intimately associated in the same cell life.

The more cells are grouped and the more highly they are specialized the more intricate become the physiological problems that present themselves. For example, if we pursue the thought that irritability is resident then it is only a step, and a short one at that, to build upon this idea a primitive nervous system. If cells can be made to change their shape and to migrate in a definite manner, it is only a thought to consider that we have two kinds of nerves, efferent and afferent, that is to say, nerves that are from and to controlling the body in question. The nerves that are spoken of may be called the sensory and the motor.

In this paper only the afferent nerves and their associate action and reflexes will be considered; as to take up the whole subject of neurology would be entirely too great a subject for one short paper. In speaking of afferent nerves, there are several different kinds with their particular and peculiar end formations which make the subject in hand very interesting and yet very difficult of thorough understanding: — for instance, the sense of sight or the sense of hearing is very difficult to describe, or understand,—the senses of smell, taste and touch are also very difficult to appreciate in their true scientific meaning.

The sense of pain does not show the simple and orderly relation between stimulus and reaction that is found present in tactile and thermal sense. Sensations of pain are set up by a heterogenous group of stimuli which do not permit of any inclusive general physical or physiological description. End-organs of the sense of pain are found to be tactile corpuscles or aborization of

* Presented at the Thirty-Ninth Annual Meeting of the Association of Surgeons of the Southern Railway System, June 16, 17 and 18, 1936

nerve endings. These are grouped differently in different parts of the body; they are found most abundant in the skin and certainly more numerous in some part of the skin than others, for instance in the palms of the hands and soles of the feet, they are very numerous, while upon the extensor surfaces of the arms and the legs and especially on the back they are sparse.

The senses of touch, pressure and pain, also the sense of temperature are very closely related. The senses of sight and hearing depend upon highly specialized nerve endings, with nerves running therefrom to the centers of appreciation and memory. The sense of taste and the sense of smell also depend upon highly specialized nerve endings, but the senses of touch, of temperature and of pressure are more simple than the mechanism previously described.

This paper is further limited to the sense of touch and closely kindred sensations, such as are transferred from place to place and recorded amid our memories. Common sensations are the relative senses, and they may be called the outstanding feature of the nervous system. Now the sense of touch may be pleasing or displeasing, depending upon its reception and association in some particular area of the brain. Now the pleasing sensation may become so exaggerated as to be displeasing, and vice versa. In one case we have the condition of pleasing,—becoming displeasing, —well—being becoming painful.

Pain may be defined as the displeasing result of an excessive stimulation of any of the nerve endings which are concerned in giving rise to sensations. Pain may also be defined as the result of stimulating nerve trunks whose sensations are recorded in the brain as displeasing. Such sensations as are received and registered as being harmful and when harmful from any cause suggest a removal either directly or indirectly. If such removal is to be had, it must come through other structures; in other words,—the information must be carried from the point of contact to the center of appreciation which must be in some bundle of neurons or in the cortex of the brain. The shortest path for an afferent sensation to travel is to the nearest neuron and from there the quickest action is obtained through an efferent nerve which goes near or to the original point of stimulation in which case we have a movement without knowledge or consent; it simply consists of an efferent nerve—a neuron—an interpolation fiber—a motor neuron—an afferent nerve and a muscle body. This in short is the modus operandi of reflex action. All this can be carried out without pain; for example when the spinal cord is severed this reflex action may be completed without the individual knowing it has taken place, but while this action in itself is complete, yet when the efferent nerve carries the impulse to the spinal cord it also carries the impulse to the basal ganglia of the brain, which in turn refers the impulse to the cortex of the brain and it is there that we receive the sensation of pain.

It all seems simple enough, but when you analyze and apply your scientific knowledge you find that it is very very difficult and you also find that there are various other centers in the brain which receive cognizance of just what is going on,—and that they too,—in sympathy with the act, behave in various and sundry ways—for instance, a child stepping on a tack withdraws the limb very rapidly long before he can think of it, his lachrymal glands become fiercely stimulated and his organs of speech are much disturbed and his eye sight is keen to appreciate just what has taken place and gives rise in the brain to a sensation described as fear and so we have introduced into the picture not only a simple reflex action, but a more complicated one suggesting pain and still more complicated by suggesting fear, all of which tends to show that the original proposition is not so simple.

The great anatomical and physiological stumbling block in the medical profession is and always has been and it seems it always will be the nervous system. While any of our reflex actions are simple and easily understood, yet there are thousands of very complicated and associated reflexes that are as yet wholly inexplainable by the most scientific in our profession.

The Purpose of Pain

IT IS conceded that the purpose of pain is to give warning that something detrimental or harmful is taking place in the body. It often so happens that pain is a warning, and the more severe pain the more definite warning, that something is happening or about to happen which will impair or destroy the body itself. A displeasing sensation which is registered in the brain, calls into action a timely movement and a reference to the intellectual centers for further action. These warnings at first may be almost negligible or they may increase in intensity until they are commanding; at all times, however, they must be considered protective.

In reference to the cerebro-spinal system, that is the different parts of the brain, the 31 pairs of spinal nerves with their ganglia are almost universally concerned in giving evidence of conditions that are displeasing or harmful to the body. The sense of touch is so delicately attuned that without the sense of sight or hearing we are able to determine accurately our immediate relation to all other bodies. Our sense of touch being grossly applied gives us a sense of pressure which may be active or passive, which when long continued or extreme may cause pain. Temperatures may be pleasing or displeasing, depending upon intensity and when they are abnormally high or abnormally low very definitely produce pain. Under this heading there may be different shades and degrees and variations of temperature which range from the abnormally low to the abnormally high, and while within certain ranges they may be pleasant, yet, if very low or very high, may become exceedingly painful. It is well known that very low or very high temperatures applied to the body will cause death of a local part. It is well known that the nervous system so acutely reacts from temperature changes that bodily harm seldom occurs. It is also well known that the sense of touch when grossly applied may be called pressure and that pressure of great intensity will cause pain and this pain is of such character that we seek relief of the pressure and thereby escape a physical damage,—it is all a matter of protection.

Most people applying to a physician or surgeon come with the expectation of getting relief from pain. Were it not for pain they would not seek relief, it is pain and the continuance of pain that causes them to seek the advice of a physician

or surgeon. Now pain is a purely suggestive symptom, its intensity must be estimated by the statements of the sufferer, by the manifestations of its presence or by any lesion which may be discovered as its possible cause. Variations in pain, are very great, and racial as well as individual. The congenitally neurotic patient will complain bitterly of pain from a cause which will only give rise to simple discomfort, in another whose nervous system is normal, it, however, should be remembered that the pain suffered by the abnormally sensitive is just as real as the slight discomfort felt from the same cause by those with normal nervous systems.

The character of pain varies in intensity from sharp and acute, to dull and aching. Acute pains are characteristic of acute inflammations or recent injuries. Acute pains of radiating character are characteristic of neuralgias or hidden organs. Dull pain is such a pain as usually attends parenchynatous viscera and many other chronic inflammations. Paraxysmal pains are the pains that are characteristic of colic and obstructions. Shifting pains are such pains as occur in the different types of rheumatism. Boring pains are such as are encountered in the spinal column and periosteal inflammations. Cramping pain is a sudden spasm of muscle groups and often cannot be distinguished from colicy pains. Cramping pains and colicy pains without differentiation are often the accompaniment of flatulence and gastro-intestinal disease. Such pains are described also in intestinal obstruction and as the result of intestinal poisoning. Other pains may be described as burning, throbbing or pulsating in rhythm with the heart's action or tenesmic, that which attends urination or defacation from an inflamed bladder or rectum. Or bearing down pains which are accompanied with a sensation of straining such as are encountered in obstetrical work. There are many adjectives describing the character of pain, but probably the ones enumerated are sufficient to cover the whole realm of thought.

As a general rule the seat of pain corresponds to the location of the causative lesion.

Vaginal Hysterectomy

Its Indications, Advantages, Technique

By Samuel Orr Black, M.D., F.A.C.S.

IN THE summer of 1877 the Medical Department of the University of Pennsylvania graduated Joseph E. Price. This young man was a Virginian by birth. In the fall of that same year, he became connected with the Philadelphia Dispensary, whose function it was to aid in caring for the health of the poor of that great city.

In a comparatively short time, Dr. Price was given control of the obstetrical department of the dispensary. Soon thereafter he introduced the department of gynecology. The bulk of his early abdominal sections were performed in the tenement rooms in the very slums themselves. It is a matter of record that he performed 100 consecutive laparotomies in that environment without a death.

So masterful was this young man's surgical technique and so well did he write and teach, that almost before he knew it, he had become a national figure in American surgery.

At the height of his career, it is said that Dr. Price operated in more cities, in more different states than has any other man before or since his time. Among the many others who flocked to Philadelphia in those early days of aseptic surgery to see Price operate, was my own father, who was just beginning to do pioneer surgery in his own home town and country. So enthused did father become over what he saw and heard that he made repeated trips to Price's clinic, only to return home and attack afresh the surgical problems as he found them, first in his own practice, and later, through consultation, in the practice of other physicians.

The older surgeons living today state the Price was without an equal in repairing the torn perineum and in performing and popularizing the vaginal hysterectomy operation, to which I now invite your attention.

Price did this operation with the Pryor clamp, often within two, three or four minutes of actual operating time, and rarely, if ever, did he use ligatures of any sort to control bleeding. He encircled the cervix with an incision through the mucosa, then pushed the vaginal wall upwards, exposing the peritoneum behind; this he opened, leading him directly into the posterior cul de sac. Through this opening, he delivered the fundus and immediately carried the middle finger of his left hand up over the posterior aspect of the fundus and forward toward the cervix, thus easily identifying the anterior or vesico-uterine peritoneum, and at the same time isolating the bladder. The anterior peritoneal fold was incised, the broad ligaments were made taut, the long blades of the powreful Pryor clamp were applied first on one side and then on the other, and with two sharp swift thrusts of the scalpel, the uterus was entirely detached and removed. Drainage was inserted between the forceps which were left in situ, and the operation was completed.

In our own practice, we have used this technique for almost every conceivable indication with the most satisfactory results.

In addition to the foregoing, we have on occasions also used the technique of Charles H. Mayo, using hemostats and then approximating the broad ligaments behind the forceps with mattress sutures of double chromic catgut, and controlling oozing and hemorrhage by means of ordinary ligature.

Again we have resorted to the technique of controlling all bleeding by suture ligature first on one side and then the other of the uterus as the uterus was pulled down into view, after pushing upwards the vaginal wall from the uterus itself. This is the method outlined by Babcock. To us, it was more tedious, requiring a longer length of operating time, had a much smaller visible operating field, and was a bit more bloody as a rule.

In the main, however, we have always turned back to the original Price operation and it is the one we now almost universally use.

Vaginal hysterectomy has the lowest operating mortality, the least postoperative ill effects and as broad a field of usefulness as any one operation with which I have any acquaintance.

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* From the Mary Black Clinic, Spartanburg, S. C.; read before the Thirty-Ninth Annual Meeting of the Association of Surgeons of the Southern Railway System, June 16, 17 and 18, 1936.

It is the one surgical procedure, apparently slighted, in all our large medical centers and surgical clinics. The operation, for some unknown reason, apparently does not enjoy the popularity it so richly deserves.

In many instances, it is very very easy to perform, in others it is sometimes extremely difficult. It can often be performed in from three to five minutes of actual operating time. It causes little if any shock, and is therefore, suitable is cases of hyperextension, myocardial or nephritic disease.

It requires but a minimum of ether if such be used as the anaesthetizing agent. It is a particular field for spinal anaesthesia. In the vast majority of cases when using clamps, not a single ligature or suture is required.

We have never had a case of pulmonary embolism or of postoperative intestinal obstruction or of femoral phlebitis to follow the use of the clamp operation. Dr. J. W. Kennedy, successor to Dr. Price, informed me that they had never had a sudden death to follow in the several thousand clamp operations they had performed.

The Pryor clamps are rather large and the jaws are thick and powerful. The handles are detachable; the clamps are easy to apply, and easier still to remove. Upon completion of the operation, when properly placed, the stumps of the broad ligaments are carried high up into the pelvis. They minimize the danger of hemorrhage. The clamps themselves act as additional drainage agents, thereby reducing the danger of infection.

Among the more important indications for its use, as outlined by Kennedy are the following:

1. Maligancy of the uterus.
2. Benign tumors, small enough to come through the pubic outlet.
3. Procidentia or marked prolapse of the functionless uterus.
4. Eroded and everted cervical lips with hyperplasia.
5. Extensive and destructive lacerations of the cervix in patients passing 40 years of age.
6. All other suspicious cases in which cancer may be lurking.
7. Hyperplastic or fibrotic uteri without tumor, having as the most common symptom, about the time of the menopause, excessive hemorrhage.

Certain cases of cancer of the fundus of the uterus are better treated surgically by vaginal hysterectomy with clamps than by any other technique with which we are familiar. Its simplicity alone makes it far superior to the Wertheim operation, which at best is long, tedious, difficult and dangerous.

Fundal carcinoma, as a rule, occurs 10 years later in life than does carcinoma of the external or internal os, and this age incidence in itself warrants less operative time and less operative shock.

As a special precaution after the patient has been placed in the proper position and the parts thoroughly cleansed, we insert through the cervix and up into the fundus a piece of gauze thoroughly wrung out of a solution of zinc chloride to prevent dissemination of malignant cells.

Cases of cervical carcinoma and of carcinoma of the middle third of the uterine body, not only occur earlier in life than do the fundal cases, but they also tend to break down quickly, to invade adjacent tissues and to metastasize early.

They nearly always occur in women who have had children and whose cervices were badly torn and left unrepaired. Cervical cancer is to fundal cancer in about the proportions of 10 to one.

Cancer of the internal os is more inaccessible and hence more diffcult to recognize early. Its close proximity to the broad ligaments makes for an early invasion of these structures and this wide spread involvement makes the prognosis grave. In this locality carcinoma is more fatal than in any other locality in the uterus.

It is only in the very earliest of cases of carcinoma of internal and external os that we now attempt vaginal hysterectomy, our method of choice being through cauterization with a large old fashioned soldering iron, followed by the application of radium and x-ray therapy.

Women, suffering from excessive uterine flow, with a uterus which is firm or hard but slightly if any enlarged, smooth in contour and outline, without and definite tumor palpable, and who are approaching or in the cancer age, have what we call a fibrotic or hyperplastic uterus and in these cases vaginal hysterectomy is one par exellence.

Uterine fibroids in women who have had a number of children and whose pelvic measurements are large and whose vaginal outlets are voluminous, so to speak, can often times be easily removed when as large as grapefruits. By resorting to morcellation, tumors much larger can be removed with surprising little hemorrhage.

There is another type of patient in which vaginal hysterectomy for a small or moderately enlarged uterus with excessive hemorrhage is especially indicated. I refer to that type of individual who usually stands surgery poorly, whose abdominal wall is prone to fatty necrosis with postoperative infection, who is apt to pick up a postoperative pneumonia or pulmonary fatty embolism, the patient whom no surgeon especially like to operate, that is the large fat woman.

Except in young women still in the child bearing age, we now rarely advise amputation for a cervical laceration with erosion or eversion. Vaginal hysterectomy is the procedure of choice in the vast majority of those cases when past the child bearing age.

There is no field in which vaginal hysterectomy has a greater usefulness than in the woman with procidentia, complete or incomplete. There are many women with their uterus protruding for a varying length through the vagina, or even hanging between the thighs with or without ulceration, infection or malignancy. In such cases the operation with clamps should have its greatest popularity. Upon completion of the operation the clamps with the broad ligaments in their jaws are carried high up into the pelvis. After the clamp is removed, each ligament retracts laterally, leaving a space which fills in with fibrous tissue thus "coalecsing the vaginal fornix with the extremities of the retracted ligaments."

In the above type of case, whenever accompanied by cystocele or retocele, hysterectomy in itself is insufficient and the operation is not complete, until a high anterior or posterior perineorrhaphy is performed at from 18 to 20 days after the primary hysterectomy. In no case is the patient permitted to be up prior to the perineorrhaphy.

Primary hemorrhage is prohibited by the use of the clamp. Secondary hemorrhage may occur here, as elsewhere, at from 10 to 14 days. Should

:t ensue, the treatment consists solely of a hypo-dermic of morphine and elevation of the foot of the bed.

A retention catheter is inserted in all our cases. It is left in situ until the day following the removal of the clamps. The bladder is irrigated with warm boric acid solution, and one to two ounces of argyrol 20% solution is instilled just prior to withdrawal of the catheter.

Forty-eight hours after operation, the clamps are opened. They are left in this condition another eight to 10 hours, at the end of which time they are simply withdrawn. The drains are removed before the clamps are opened. From here on, absolutely nothing is done to the vagina unless a little vaginal discharge develops at the end of six to eight days. Then a low warm cleansing douch is given.

The vaginal approach for surgery of the pelvis is by no means limited to the uterus itself.

The posterior uterine cul de sac is separated from the vaginal by tissues only a few millimeters in thickness. Its accessibility is, therefore, quick and safe. In suspected ruptured ectopic pregnancy this narrow partition may be opened and if rupture has occurred a gush of blood will ensue; the finger is then inserted, the tube localized, pulled down and the entire operation is over with.

Although a few surgeons operate through this route for bleeding uteri complicated by suppurative lesions of the tubes and ovaries, we mention the proceure only to condemn it. It is our belief that these lesions should be attacked from above, where we can both the better see and feel, Moreover, oftentimes a portion of the small intestine is down in the pelvis, fastened more or less tight and unless we relieve that attachment, postoperative obstruction may take place. We firmly believe that the surgical toilet should be complete, all the pathology removed, the small bowel brought entirely up out of the pelvis and the large bowel placed in its anatomical position and the small bowel carefully walled off from the site of the pre-existing pathology either by means of the omental or by the careful introduction of plenty of rubber covered drains forming a partition across the entire pelvis, for by this means and this alone, can we reduce to a minimum the danger of obstruction, as well as of peritonitis, and offer to the patient her best chance of a prompt and uninterrupted convalescence.

In conclusion we feel that vaginal hysterectomy with clamps is a procedure which has not been awarded its proper place in surgery.

Postoperative hemorrhages, postoperative obstruction, postoperative shock and cases of pulmonary embolism are all but unheard of after this operation.

Therefore, as the postoperative effects are all but nil, and as the time of the operation is reduced to a minimum, often to but three to five minutes, and as the mortality rate is so low as to be comparable to that of radium without the "sequellae and relapses" of the latter, and as the operation as pointed out by J. W. Kennedy violates no single surgical principle, we have no hesitancy in recommending it for your earnest and careful consideration, with the full expectation that few trials on comparatively easy cases to begin with, will at once prove its merit and warrant a more generous use at your hands.

The Treatment of Fractures

Report of Several Thousand Cases

By Edward Thomas Newell, M.D., F.A.C.S.,
Chattanooga, Tennessee

AT THE March, 1934, meeting of the Southeastern Surgical Congress, Nashville, Tennessee, I presented a paper on "The Fracture Problem" with a study of 10,082 fractures treated at the Newell Clinic, 1908 to 1934. Since January, 1934, we have treated at the Newell Clinic 918 fractures.* The total from 1908 to May 31, 1936, was 10,971 cases as follows:

Site	Number
Head	700
Spine	246
Chest	984
Pelvis	370
Clavicle	295
Scapula	90
Humerus	240
Elbow	314
Radius	1141
Ulna	677
Hand	2182
Femur	363
Patella	61
Tibia and fibula	1272
Foot	2036
Total	10,971

Fractures treated at the Newell Clinic during the past 28 years up to May 31, 1936.

In interpreting these figures you will see that fractures of the head, fractures of the spine, and fractures of the pelvis are much more frequent than before the advent of the automobile, airplane, motorcycle and the ever-present, huge, high-powered machinery of the present day.

Fractures of the radius (Colle's) is the most frequent fracture in the body and accounts for more than 10% of the fractures in the series.

There were 206 open operations (not including operation on compound fractures) done on these 10,971 fracture cases. This is a little less than 2% of cases subjected to open operation.

The 206 operations were done (not including compound fracture operations) on the following bones—up to May 31, 1936:

Clavicle	4
Humerous	8
Elbow	15
Femur	11
Radius and Ulna	53
Tibia and Fibula	31
Patella	50
Maxilla	13
Depressed Zygomatic Arch	19
Ribs	2

Attention is called to 50 operations on the patella, which are 25% of all the open operations performed; and to 19 open operations done on depressed zygomas, 10%. We consider open operation the method of choice in the treatment of the above fractures.

The Railway Surgeon in any small town on his railroad is apt to have one or more serious fractures brought to him at any time, and he should be acquainted with the best authenticated methods of first aid and treatment. He should have at his

* Presented at the Thirty-Ninth Annual Meeting of the Association of Surgeons of the Southern Railway System, June 16, 17 and 18, 1936.

command adequate equipment for first aid and for treatment, and should further be able to select the cases for complete treatment under his supervision, and those to be referred for treatment to the specialist and for hospitalization.

In a recent address before the Southeastern Surgical Congress, I had the following to say in regard to "The Fracture Problem:"

"It is worthy of note that in the past 25 years more progress has been made in the treatment of fractures than was made in the previous century. We have made many changes in our treatment in the past 25 years in fracture work. As time goes on, with improvement in technic and equipment, we will no doubt make other material changes.

"The proper management of a fracture begins 'where and when it occurs'—especially in fractures of the extremities. Ambulance men, first aid men in railroad shops and factories, and designated filling stations should be trained in the use of the Thomas splints or its modifications. The slogan 'splint them where they lie' is most expressive and often prevents a simple fracture from becoming compounded, inhibiting further trauma to soft parts, muscles, vessels and nerves.

"Efficient first aid and proper transportation to the doctor's office or the hospital, aids materially in the satisfactory examination and diagnosis of the fracture. In severe trauma, a hypodermic of morphine quiets the patient and tends to overcome the muscle contractures that always manifest themselves in a major fracture, and increase deformity and trauma. The extremity can, in the first aid splint, be placed in the proper positions for correct x-ray interpretation by films or fluoroscope, without producing further pain, deformity or injury to the soft parts.

"All known fractures, as well as suspected fractures, should be x-rayed in the classic positions before any treatment is attempted. As multiple fractures are so frequently encountered today in serious injuries, the free use of the x-ray is advisable at all possible fracture sites. Negative x-rays are comforting to the patients and are of value to the physician and corporation.

"There are three essential factors in the order named to be considered in the treatment of serious fractures:

1. The life of the patient (shock—hemorrhage—internal injuries).
2. The extent and location of the injury to the soft parts.
3. The bone or bones fractured, (site, character of fracture-transverse, oblique, spiral, comminuted or compounded—if infected).

"I regret to say that too frequently in the past, treatment has been primarily directed only to the bone lesion. No greater error could be committed."

Diagnosis

THE diagnosis of a fracture is made with x-ray films, A.P., lateral, oblique, fluoroscopic, and clinically. The chief reliance in the diagnosis of a fracture is dependent upon the x-ray findings. The fluoroscope is of inestimable value in the setting of a fracture, but should not be relied upon for diagnosis; for in a minute fracture, it might easily lead you into error. The portable x-ray of the present day, which is shock proof, with its extreme flexibility can be placed in almost any position. This, together with the new and better vison fluoroscope, adds a very great deal to the present day advanced treatment of fractures.

Treatment

FOR relaxation, general anesthesia (preferably ether), local anesthesia in selected cases. When one realizes that there are 1,000,000 fractures to be cared for annually in the United States occurring under any and all conditions, you can readily understand the difficulty of a "standardised treatment" that would be applicable to all cases. Yet, we must have a general working plan for the rendition of first aid and later for efficient treatment.

The proper application of extension in extremity fractures, in the Murry Jones or Keller-Blake splints will accomplish the former, and the proper splinting (Thomas splint or plaster) after the setting of the fracture, should accomplish the latter.

Where there is great trauma to the soft parts, they should always receive first attention; later the bones can be set.

In approximating fragment 100% approximation while desirable is not absolutely necessary or essential, but 100% alignment should be the fracture surgeon's goal.

As for splints, the Thomas splint or its modification is most suitable for fractures of the lower extremity, when indicated. Some lower extremity fractures are best treated with plaster. The Whitman method in fractures of the neck of the femur is exceedingly popular with orthopedic surgeons and has a very definite place.

Fractures in the lower third of the leg, both bones where they can be held in proper approximation and alignment, are best treated with plaster; (circular that has been bi-valved).

A great many fractures of the lower extremities, oblique and spiral in character, cannot be satisfactorily treated by ordinary skin traction. They require either the Ransohoff tongs or some of its modifications; the Steinman pin, or better the application of the Kirschner wire with or without the assistance of the Hawley or Albee table.

Fractures of the upper extremity of the humerus are best treated when oblique or spiral, by extension in the Murry Jones splint, and later they should be put up in an airplane splint either at an acute or a right angle.

It is best to secure these splints to the body with plaster, and also to incorporate the shoulder and forearm in plaster. There should be traction on the fracture line with an adjustable screw which regulates the amount of traction necessary to correct overriding or angulation, at the fracture site.

Colle's fractures are best treated with anterior and posterior molded plaster of Paris splints. These splints can be removed and the forearm massaged and the wrist joint mobilized early, which is a distinct advantage in the recovery of function.

Lately it has been the custom of a large number of orthopedic surgeons and fracture surgeons to apply, where there is not much swelling and no trauma, plaster of Paris directly to the skin. It is claimed that it holds the fragments in better apposition and produces no ill effects. In well selected cases and in the hands of competent orthopedic and fracture surgeons this procedure may be carried out with good results and no morbidity, but the *general use* of such a plan of action, in the

treatment of fractures, would result disastrously to both the surgeon and the patient. Already as a result of the injudicious use of plaster in this manner, there are now many pressure ulcers and necrosed tissue areas, throughout the country.

I am afraid that we are going to find in addition, more Volkmanns contractures (a hideous spectacle), if this innovation in plaster application becomes more general.

Personally I see no real occasion for putting plaster directly on the skin, and I think that Railway and Emergency Surgeons, as a whole would do well to continue to have their splints bi-valved and well-padded.

In conclusion we feel that most of the equipment used in the treatment of fractures should be made by the surgeon doing the work rather than purchased from splint and appliance makers.

Meckel's Diverticulum

By Albert C. Jackson, M.D., F.A.C.S.,
Jasper, Alabama

THIS congenital anomaly exists because of failure to close of a portion of the omphalomesenteric or vitelline duct.* Investigations have shown that this duct should close in the fetus before the fourth month. A partial description of this abnormality was first recorded by Littre in 1770. The credit for the discovery of its origin goes to Johann Frederick Meckel who gave a full treatise on the subject in 1808.

It exists as a blind pouch off the small intestine opposite the mesenteric attachment somewhere in the last 18 inches of the ileum in children, however, in adults, it may be located as far as three feet from the ileocecal valve. It differs from other diverticula in that it is always single and has all the coverings represented in the normal intestine in an attenuated form and except in the rare instance where one grows between the mesenteric folds is always located opposite the mesenteirc attachment. The condition has been estimated to exist in from 2% to 3% of all people and predominates by about five to one in males.

It may exist in many different forms and shapes and has been classified by Christie into five different types with percentages found from a study of 63 autopsy specimens. There were 82.5% of the typical diverticulum ending in a blind pouch and floating free in peritoneum cavity, 10% with partial obliteration and fibrous cord attached to the umbilicus, another portion of the intestine, the anterior or posterior abdominal wall, or in a hernia, and 6.3% with the umbilical fistula. The more rare forms are the extremely large irregular shaped diverticulum growing between the folds of the mesentery, and the umbilical polyps. A Meckel's diverticulum may have its own mesenteric attachment and blood supply, or may get its blood supply from the intestinal mesentery. It ranges in size from a small blind pouch ½ inch long to an extensive tube as in the case reported by Moll II which measured 33½ inches in length.

Histologically the diverticulum presents all the normal layers of the intestinal wall in a rudimentary form and the lining very often shows typical gastric mucosa, aberrant pancreatic tissue or a mixture of both. Rarely, tumors either benign or malignant, may be found in a diverticulum.

The fact that an individual may have a diverticulum during his entire life without causing symptoms and incidentally at autopsy it is found to be free from adhesions or other signs of inflammation, leads me to say that there are no symptoms unless some of the complications develop. Therefore, a diagnosis is seldom made. Among some of the complications are intestinal obstruction either partial or complete, brought on by kinks, volvulus, or intussusception, diverticulitis with peritonitis, gangrene of the diverticulum as of the appendix, peptic ulcer on the mucosa of the diverticulum with or without perforation, and tumor formation in the diverticulum with or without obstruction. These complications show up as a rule early in life or it may be the 3rd or 4th decade before they are in evidence.

Those cases that develop intestinal obstruction have the classical symptoms of this condition and are unlike intussusception in that there is no palpable tumor, and there is no mucous and blood in the stools showing up after the other symptoms are in evidence. If there is blood in the stools it is not mixed with mucous and has been present before other signs appeared. In the cases developing inflammation and gangrene the symptoms are almost identical with appendicitis and this mistaken diagnosis is almost always made, since of all the acute conditions of the abdomen, appendicitis is by far the most common.

A brilliant chapter has been added to this subject by numerous investigators in the past 30 years and a collection of 33 cases has been reported of ulcer on the mucosa of Meckel's diverticulum, many of which have perforated. The ulcers almost invariably occur at the line of junction of the aberrant gastric mucosa of the diverticulum and the intestinal mucosa very similar to a gastrojejunal ulcer following gastro-enterostomy. The ulceration point is where the acid secretion from the gastric mucosa in the diverticulum comes in contact with the alkaline secretion of the intestinal mucosa. The ulcer is apparently due to digestion of the intestinal mucosa by the highly acid secretion, has the appearance of a peptic ulcer and tends to bleed and finally to perforate.

The outstanding symtoms of this condition are paroxysmal pain, and recurrent intestinal hemorrhages, and characteristically occurring about five to one as often in males as in females. When a perforation occurs, there is a sudden sharp pain in the abdomen near the umbilicus which can rarely be controlled by morphine, followed by a rise in temperature, pulse, and white blood count with vomiting. There is board like rigidity of the abdominal muscles more marked on the right side and considerable tenderness on pressure in the para-umbilical region mostly to the right side. Patients presenting these symptoms with a previous history of intermittent intestinal hemorrhage should be diagnosed perforated diverticular ulcer and advised to have immediate operation and all cases of intermittent intestinal hemorrhage of unexplained origin should be advised to have exploratory laparotomy in the hope that a bleeding diverticular ulcer may be found and given the proper attention before it perforates.

The most rare complication of Meckel's diverticulum is a tumor formation which may be evidenced by intestinal hemorrhage and a tumor mass in the para-umbilical region. They should

* Presented at the Thirty-Ninth Annual Meeting of the Asosciation of Surgeons of the Southern Railway System, Knoxville, Tenn., June 16-18, 1936.

have operation with resection of a portion of the intestine in most cases.

The treatment is entirely surgical and since such a variety of conditions may be found we should be prepared to do anything from a simple resection of the diverticulum with closure, to a complete resection of the intestine with anastamosis. In the desperately ill patients with intestinal obstruction from kinks, bands, or volvulus it may be best to relieve the obstruction and leave the diverticulum alone to be cared for at a later operation. When a simple resection of the diverticulum with a closure of the opening in the intestine is done, it is well to remember that your sutures should be placed with the long axis of the intestine so as not to produce a narrowing of the lumen.

In my practice during the past 16 years, I have seen six cases, four males and two females.

The first case was a male child eight months old. Diagnosis intestinal obstruction. Operation, simple resection of diverticulum with closure after volvulus was relieved. Uneventful recovery.

CASE No. 2. White boy 14 years of age. Diagnosis, intestinal obstruction. Operation, diverticulum with fibrous cord attached to umbilicus and a loop of intestine kinked over this cord. The hand was cut loose and the diverticulum resected with simple closure. Uneventful recovery.

CASE No. 3. White male, age 8 years. Diagnosis, intestinal obstruction. Operation revealed diverticulum buried in adhesions partially gangrenous, attached to posterior abdominal wall, with partial obstruction. Dissection of adhesions, resection of diverticulum with simple closure. Considerable distention with nausea and vomiting after operation but made a complete recovery.

CASE No. 4. White male, age 6 years. Diagnosis, acute appendicitis. Operation, appendix shows slight inflammation. Exploration revealed a simple diverticulum inflamed but floating free which was resected and the opening closed. He made an uneventful recovery.

CASE No. 5. White girl, age 15 years. Diagnosis, appendicitis or Meckel's diverticulum with partial obstruction. Operation, inflamed appendix removed. Exploration revealed about eight inches of the ileum bound with dense adhesions to the posterior abdominal wall with partial obstruction. When the adhesions were dissected and the intestine made free, a small diverticulum was found which was resected and the opening closed. She had a very stormy recovery going through all the stages of general peritonitis before showing signs of improvement. Now in good health.

CASE No. 6. White female, married, mother of two children, age 22, now pregnant eight months. Twelve hours before admission developed sudden acute, excruciating pain just to the right of the umbilicus, followed by nausea and vomiting. Morphine was given in repeated doses with no relief, and the pain seems to grow worse. Past history was that she had experienced two or three attacks of abdominal pain in the past two months, not so severe. She had not had any intestinal hemorrhages. She was admitted to the hospital on December 29, 1932, suffering acute abdominal pain and vomiting. Examination shows well developed and nourished white female, pulse 114, temperature 101, respiration 22, total white blood count 16,750. Heart and lungs normal. Abdomen presents a gravid utuerus near term, muscles very rigid and more marked on the right side, marked tenderness on pressure about three inches below the tip of the ninth right costal cartilage. A dianosis of acute perforating appendicitis was made and a laporatomy done. Free pus with general peritonitis was found. The appendix was acutely inflamed and removed. Not satisfied that the appendix could cause such diffuse peritonitis, a further search was made and a sausage-shaped inflammatory tumor of the ileum about 18 inches from the ileocecal valve was delivered. After some dissection a Meckel's diverticulum was discovered plastered to the side of the intestinal wall by adhesions and at its base there was a small punched out perforation large enough to admit a match stem and presenting the exact picture of a perforated peptic ulcer at the pylorus. There was so much inflammation with thickening and induration of the intestinal wall that a simple resection of the diverticulum would not suffice in this case. About eight inches of the intestine was resected and an end to end anastamosis done. The following day her condition was fair except for considerable distention. Thirty six hours after operation she went into labor and delivered. Following this she made a very nice recovery and was discharged from the hospital 14 days after admission in splendid condition. She is in good health today. Pathological report showed a typical gastric mucosa in the divertulum.

J. M. Mason, in an analysis of 33 reported cases showing ulcers of Meckel's diverticulum, pointed out that hemorrhage was the outstanding symptom in 32 and was not mentioned in one. Perforation occurred in 14 of the 33 cases. My case denied having intestinal hemorrhage, and since her hemoglobin was 80, the total red count was 4,125,000, there was no evidence of hemorrhage. She admitted having a few attacks of abdominal pain. It is my opinion that with further study and report of cases of diverticular ulcer, it will be shown that there are many cases of ulcer without bleeding.

A study of the literature on this subject shows that one medical man who referred a case to a surgeon and observed the findings at operation, seven years later referred another case correctly diagnosed, and this is a fine example for all of us to emulate. Unexplained intestinal hemorrhage in any patient early in life should make us think of diverticular ulcer and advise an operation before perforation. During every exploratory laporotomy where there is intestinal hemmorhage or peritonitis, a search for a Meckel's diverticulum should be made. In many cases when a diagnosis of appendicitis has been made and the pathology of the appendix does not explain the symptoms, a diverticulum should be suspected and sought for. Abdomens have been closed and a perforated diverticular ulcer found at autopsy. Since we have been given so much illuminating literature on this subject during the past 25 years, it is now time for us to begin diagnosing it correctly, advising operation before perforation occurs, and really exploring an abdomen when it should be done.

Owing to the fact that appendicitis is most often the mistaken diagnosis made in perforated diverticular ulcer is, in my opinion, a strong argument for immediate operation in the cases with an acute surgical abdomen, instead of watchful waiting as advocated by some.

Association Meetings

—Programs of Special Interest—

American Association

THE Twenty-First Annual Meeting of the American Association of Industrial Physicians and Surgeons will be held at the Traymore Hotel, Atlantic City, N. J., on Wednesday, Thursday and Friday, October 7, 8 and 9, 1936. The program is as follows:

Wednesday Afternoon, October 7

Chairman, Dr. Loyal A. Shoudy, Chief Surgeon, Medical Dept., Bethlehem Steel Co., Bethlehem, Pennsylvania.

2:00—Teaching First Aid Instructors, by Dr. Harry W. Gentles, B.C.M., Director First Aid Dept., Chicago Chapter, American Red Cross. (The important difference between teaching first aid to students and teaching first aid to instructors. A resume of the qualifications of a good first aid instructor. How he should be taught to teach. How he can assist in preventing accidents.)

2:30—Discussion.

2:45—Methods of Handling the Injured Worker to Get Him Back on the Job, by Dr. Niel E. Eckelberry, Surgeon in Charge, Medical Dept., Consolidated Edison Co., Inc., and Affiliated Companies, New York City. (Why it is good practice to get the injured worker back on the job as soon as possible. The methods commonly followed. Important factors.)

3:15—Discussion.

3:30—What Remedies May Be Applied to Minimize Accidents Due to Physical Deficiencies, by Dr. A. F. Lecklider, Personnel Director, Fisher Body, Detroit Division, General Motors Corp., Detroit, Mich. (Are men with physical deficiencies more prone to accidents? Should such men be permitted to operate mechanical equipment? What part do physical examinations play in determining the proper remedies?)

4:00—Discussion.

Thursday Morning, October 8

Chairman, Dr. Floyd E. Shaffer, President, A. A. I. P. & S.

9:00—Business Meeting.

9:45—Trends of Industrial Hygiene with Particular References to the Prevention of Occupational Diseases, by Dr. Clarence D. Selby, Medical Consultant, General Motors Corporation, Detroit, Mich.

Discussion opened by Dr. G. H. Gehrmann, Medical Director, E. I. Du Pont de Nemours & Co., Wilmington, Delaware.

The Employability of the Arrested Case of Tuberculosis, by Dr. B. L. Vosburgh, General Electric Co., Schenectady, N. Y.

The Relation of Industrial Life to Gastro-Intestinal Disease, by Dr. Burrill B. Crohn, Gastro-enterologist, New York City.

Thursday Afternoon

Round Table Discussion led by Dr. A. J. Lanza, Assistant Medical Director, Metropolitan Life Insurance Company, New York City.

Report of Nominating Committee and Election of Officers.

Thursday Evening, Banquet, 6:30 P.M.

Program to be announced.
Installation of Officers.

Friday Morning, October 9, Occupational Diseases

Chairman, Dr. Hart E. Fisher, Vice-President for Health, National Safety Council, and Chief Surgeon, Chicago Rapid Transit Co., Chicago.

9:45—The Lesser Known Facts About Common Occupational Diseases, by Dr. Robert B. Hunt, Medical Adviser, American Mutual Liability Insurance Co., Boston, Mass. (Facts given in the language of the layman, not saturated with medical terms, of many of the occupational disease hazards, treatment, recurrence, and other important phases.)

10:15—Discussion.

10:30—The Relation of Pre-employment and Periodic Physical Examinations to Occupational Disease Hazards, by Dr. W. C. Templer, Medical Director, Corning Glass Works, Corning, N. Y. (The various steps of pre-employment and periodical physical examinations. The various defects found and their relation to occupational disease hazards. How such examinations help all concerned.)

11:00—Discussion.

11:15—What Can the Engineer Do to Eliminate the Hazards of Occupational Diseases?, by Dr. Reubel C. Stratton, C.E., Supervising Chemical Engineer, The Travelers Insurance Co., Hartford, Conn. (Important factors such as layout, ventilation, safe handling methods, and other practices often overlooked by the safety engineer. Why it is necessary to consider all operation from the storage of raw materials to the shipment of finished products.)

11:45—Discussion.

Program Committee:
W. A. Sawyer, M.D., Chairman.
McIver Woody, M.D.
E. C. Holmblad, M.D.

The dates and place of this program coincide with the dates and place of the 25th National Safety Congress, one of the Fellows of the American Association of Industrial Physicians and Surgeons, Dr. Cassius H. Watson, being President of the National Safety Council. Wednesday afternoon's meeting, presided over by Dr. Loyal A. Shoudy, is a joint meeting with the Industrial Health Section of the National Safety Council, as is also Friday morning's session on Occupational

Diseases, presided over by DR. HART E. FISHER, Vice-President for Health, of the National Safety Council.

New York Central Surgeons

THE Thirteenth Annual Meeting of the Association of Surgeons of the New York Central System will be held at the Hotel Statler, Detroit, Michigan, on Tuesday, Wednesday and Thursday, October 13, 14 and 15, 1936. The program is as follows:

Tuesday, October 13

9:00 A.M.—Registration and Information Bureau, Ballroom foyer.
Golf Tournament, Detroit Country Club (Must tee off by 3:00 P.M.).

2:00 P.M.—Ladies' Bridge-Tea, Ivory Room.

8:30 P.M.—Informal Reception to the President of the Association, in Banquet Hall, followed by dancing and refreshments.

Wednesday, October 14; Scientific Session

8:30 A.M.—Registration continued, Ballroom foyer.

9:30 General Meeting, Convention Hall—Banquet Hall on Ballroom floor.

1. Presidential Address—DR. W. H. McNEILL, JR., New York City.
2. Plastic Surgery of the Face, by DR. CLARE STRAITH, Detroit, Mich.
(Illustrated by lantern slides)
Motion Picture — Correction of Hump Nose Deformities.
3. Symposium on Hernia:
1. DR. J. G. FROST, Chicago, Ill.
2. DR. FRANK KELLY, Detroit, Mich.
3. DR. BRADLEY L. COLEY, New York City.
General Discussion.
4. Post-traumatic Periarticular Fibrosis, by DR. DONALD GORDON, New York, N. Y.
Discussants—DR. GLEN BROUGH.
DR. FRANK CURTIS.

9:30 A.M.—Oculists' Session — Buses leave Hotel Statler for Ann Arbor for the following program: Clinic by DR. FRANCIS BRUCE FRALICK at the University of Michigan. Dr. Fralick plans to give a dry clinic in the morning. Luncheon will be served at the Hospital, and DR. FRALICK will give an operative clinic in the afternoon.

Clinical Session

2:00 P.M. to 4:00 P.M.—Grace Hospital.

1. The Conserative and Operative Management of Head Injury, by DR. E. S. GURDJIAN, Dertoit, Mich.
2. Some Problems in the Handling of Fractures, by DR. FRANK CURTIS, Detroit, Mich.
3. Surgical Treatment of Tuberculosis and Its Relation to the Industrial Surgeon, by DR. W. A. HUDSON, Detroit, Mich.

7:30 P.M.—Annual Dinner, Ballroom.
FRANK V. WHITING, Toastmaster.
(Name of principal speaker to be announced)
Distribution of golf prizes.
Music, entertainment and dancing.

Thursday, October 15, Scientific Session

9:00 A.M.—1. Traumatic Shock, by DR. GEORGE H. MARCY, Buffalo, N. Y.
Discussant—DR. L. A. ENSMINGER, Indianapolis, Ind.

2. Edema, by DR. GORDON MYERS, Detroit, Mich.
General Discussion.

3. Visual Standards and Periodic Examinations, by DR. GEORGE P. MYERS, Detroit, Mich.

4. Colles' Fracture, by DR. WILLIAM C. KUNKLER, Terre Haute, Ind.
Discussant—DR. W. H. MARCY, Buffalo, N. Y.

9:30 A.M.—Oculists' Session—Parlor B. (Program under direction of DR. W. R. PARKER, assisted by DR. C. E. MCDANNALD, to be announced)

Business Session

Reports of Committees.
New Business.
Election of Officers.
Final Adjournment.

Clinical Session

2:00 P.M. to 4:00 P.M.—The Henry Ford Hospital. (Program to be announced at meeting).

DR. E. L. WHITNEY will give a clinic for oculists at the same time DR. MCCLURE is conducing the general surgical clinic.

Ohio State Medical

THE Ninetieth Annual Meeting of the Ohio State Medical Association will be held at Cleveland, on Wednesday, Thursday and Friday, October 7, 8 and 9, 1936. The program for the Section on Public Health and Preventive Medicine includes the following:

Friday, October 9

Diagnostic Methods in Industrial Medicine, by PAUL A. DAVIS, M.D., Akron.

(Differentiation between industrial medicine and general medicine. Methods of absorption of various materials used in industry. Scientific laboratory tests used in confirmation of physical findings. Dust problems, vapor problems and solvent problems. Skin reactions and differential diagnostic methods. Treatment of various industrial conditions.)

Discussion, A. G. CRANCH, M.D., Cleveland.

A Biologic Test for the Determination of the Fibrogenetic Properties of Dust, by CAREY P. MCCORD, M.D., J. A. KASPER, M.D., and W. L. BROSIUS, M.D., Detroit (by invitation).

(Despite chemical methods, x-ray examinations, and dust counts—one or all combined—it is im-

possible to have sufficient information as to the prospective damage from the dust in any given work place. As shown by Miller and Sayers, dusts injected into the peritoneal cavities of small animals bring about an essentially characteristic response which may be specified physiologically as proliferative, absorptive, or inert. These changes are quite apparent at the end of 30 days, but at the end of 60 or 90 days, the lesions produced are more easily identified. This present paper sponsors the worth of this procedure as a practical test for throwing additional light on the probable behavior of dust entering the lungs of workmen during the course of employment. Animal demonstrations.)

Discussion, W. EUGENE MASTERS, M.D., Columbus.

General discussion.

Ingestion of Carbon Tetrachloride (A Public Health Hazard), by J. J. SELMAN, M.D., and Z. T. WIRTSCHAFTER, M.D., Cleveland.

(Carbon tetra-chloride has been long known to be a hazard because of its use (1) as a solvent, (2) in fire extinguishing fluids, (3) in medical therapy as an anthelmintic. The effects of oral ingestion of carbon tetra-chloride in larger than therapeutic doses has been studied by us in two cases occurring in chronic alcoholics who drank the substance as a substitute for alcohol. The outstanding manifestations were the severe hepato-renal lesions as evidenced by severe jaundice, extreme elevation of the icteric index, marked nitrogen retention and oliguria. Corrobation by autopsy findings in one fatal case. The hepato-renal factor has not been sufficiently emphasized in the past. Carbon tetra-chloride presents a menace because it is a common household commodity, therefore, subject to accidental oral ingestion. It is, therefore, important to recognize the clinical syndrome.)

Discussion, EMERY R. HAYHURST, M.D., Columbus.

General Discussion.

New York State Society

THE Sixteenth Annual Meeting of the New York State Society of Industrial Medicine will be held at the Hotel Van Curler, Schenectady, N.Y., Thursday, November 5, 1936. DR. CHARLES W. WOODALL, of Schenectady, President of the Society, will preside. The program will start at 9:45 a.m. with a business session, followed by a scientific program as follows:

Workmen's Compensation Law as Related to Medical Practice, by DR. D. J. KALISKI, New York City, Chairman of Workmen's Compensation Committee, Medical Society of the State of New York.

Relationship of Trauma to Malignant Tumors, by FRED W. STEWART, M.D., Memorial Hospital, New York City. Discussion opened by ELLIS KELLERT, M.D., Ellis Hospital, Schenectady, N.Y.

Chronic Subdural Hematoma, by C. C. COLEMAN, M.D., Richmond, Virginia. Discussion opened by A. R. GRANT, M.D., Utica, N. Y.

Reduction of Fracture-Dislocation of the Cervical Spine with Skeletal Traction, by W. GAYLE CRUTCHFIELD, M.D., Richmond, Virginia. Discussion opened by DR. E. K. CRAVENER, Schenectady, N. Y.

At a luncheon meeting of the Board of Directors, new officers will be elected for the coming year. The usual practice of the Society is to advance the Vice-President to the Presidency. In this event, DR. WILILS C. TEMPLER of the Corning Glass Works, Corning, N. Y., will be the new President and DR. EDGAR A. VANDER VEER of Albany, N. Y., will be advanced to the 1st Vice-Presidency, a new 2nd Vice-President being elected to fill that vacancy. DR. WOODALL, as retiring President, will go on the Board of Directors as a member ex officio.

The afternoon program will be devoted to a symposium on the Treatment of Hernia, with papers as follows:

Injection Treatment of Hernia, by DR. ARTHUR F. BRATRUD, Minneapolis, Minnesota.

Hernia Treatment at Ruptured and Crippled Hospital, by DR. NORMAN L. HIGINBOTHAM, New York City.

Some Present Day Aspects of the Hernia Problem, by DR. E. MACD. STANTON, Schenectady, N. Y.

The program is open to all members of the medical profession interested, whether they be members of the Society or not, and the committee issues a cordial invitation to that effect.

Congress of Railway Surgeons

THE 47th Annual Meeting of the American Association of Railway Surgeons—convening jointly with the Association of Surgeons of the Chicago, Milwaukee, St. Paul & Pacific Railroad (Lines East), the Association of Surgeons of the Illinois Central System, the Association of Surgeons of the Chicago & North Western Railway, and the Surgeons of the Burlington Railroad —will be held at the Palmer House in Chicago, on November 5, 6 and 7, 1936, with the following program:

Thursday, November 5

9:00 to 9:45 a.m.—Registration, Visiting Exhibits.
Morning Session:
9:45 a.m.—Chicago, Milwaukee, St. Paul & Pacific Railroad:

1. General Remarks.
   Operation for Inguinal Hernia, (animated movie film), by DR. A. R. METZ, Chief Surgeon Lines East, Chicago, Ill.
2. Peroral Endoscopy, by DR. JOHN S. KNIGHT. Otolaryngologist, Kansas City, Mo.
   Discussion by DR. L. F. MCBRIDE, Chicago, Ill.
3. Fractures of the Lower End of the Radius, by DR. RALPH M. CARTER, President, Wisconsin State Medical Society, Green Bay, Wis.
   Discussion by DR. O. R. LILLIE, Milwaukee, Wis.
4. Obstruction of the Bowel Due to Lesions of the Rectum and Colon, by DR. VERNON C. DAVID, Professor of Surgery, Rush Medical College, Chicago, Ill.

Afternoon Session:
2:00 p.m.—Illinois Central System:

1. Arthritis—Personal Views with Remarks on Recent Developments, by DR. WILBER E. POST, Consulting Internist, Chicago, Ill.
   Discussion by DR. LEROY H. SLOAN, Internist-in-Charge, Chicago, Ill.

2. The Surgical Treatment of Arthritis, with lantern slide demonstration, by Dr. Philip H. Kreuscher, Consulting Surgeon, Chicago, Ill.
   Discussion by Dr. Fred W. Bailey, Consulting Surgeon, St. Louis, Mo.
3. The Treatment of Peptic Ulcer Based on Physiologic Principles, with lantern slide demonstration, by Dr. Alton Ochsner, Consulting Surgeon, New Orleans, La.
   Discussion by Dr. J. W. Barksdale, Division Surgeon, Jackson, Miss.
4. Early Pathology in Bone Tumors, with lantern slide demonstration by Dr. Robert D. Schrock, Local Surgeon, Omaha, Nebr.
   Discussion by Dr. Chester C. Guy, Adjunct Surgeon-in-Charge, Chicago, Ill.

Friday, November 6

9:00 to 9:30 a.m.—Visiting Exhibits.

Morning Session:

9:30 a.m.—Chicago Burlington & Quincy Railroad, Chicago & North Western Railroad.

1. Importance of X-Ray Examinations and Proper Interpretation, by Dr. E. L. Jenkinson, Chicago, Ill.
   Discussion by Dr. R. W. Fouts, Omaha, Nebr.
2. Abdominal Trauma and Its Diagnosis, by Dr. McMicken Hanchett, Council Bluffs, Iowa.
   Discussion by Dr. E. P. Heller, Kansas City, Mo.
3. Circulatory Diseases of the Extremities, (lantern demonstration), by Dr. Carl Johnson, Chicago, Ill.
   Discussion by Dr. Francis Murphy, Milwaukee, Wis.
4. Fractures Femurs, (Motion Pictures), by Dr. R. M. Carter, Green Bay, Wis.
   Discussion by Dr. F. M. Keefe, Clinton, Iowa.

Afternoon Session—2:00 p.m.:

1. The Care of Ocular Foreign Bodies, by Dr. S. R. Gifford, Chicago, Ill.
   Discussion by Dr. Sidney Walker, Chicago, Ill.
2. Can an Injury Cause a Malignant Tumor?, by Dr. H. H. Davis, Omaha, Nebr.
   Discussion by Dr. A. R. Metz, Chicago, Ill.
3. Early Recognition of Cardiac Insufficiency, by Dr. John C. Parsons, Creston, Iowa.
   Heart-Arterio-Renal Disease in Relation to Railroad Cases, by Dr. Francis Murphy, Milwaukee, Wis.
   Discussion by Dr. Geo. K. Fenn, Chicago, Ill.

Saturday, November 7

9:00 to 9:30 a.m.—Visiting Exhibits.

9:30 a.m.—Chicago, Burlington & Quincy Railroad, Chicago & North Western Railroad.

1. Skull Fractures, Diagnosis and Treatment, by Dr. E. C. Henry, Omaha, Nebr.
2. Treatment of Electrocuted Persons, by Dr. H. A. Brennecke, Aurora, Ill.
   Discussion by Dr. Hart E. Fisher, Chicago, Ill.
3. Conservative Treatment of Fractures by Adhering to Fundamental Principles, by Dr. J. W. Martin, Des Moines, Iowa.
   Discussion by Dr. H. W. Orr, Lincoln, Nebr.
4. Fractures of the Os Calcis, by Dr. M. S. Henderson, Rochester, Minn.
   Discussion by Dr. P. E. Sawyer, Sioux City, Iowa.

American College of Surgeons

THE 26th Annual Congress of the American College of Surgeons will be held in Philadelphia, Ocotober 19-23, 1936. On Wednesday, October 21, at 2:30 P.M., there will be a conference, or symposium, on industrial medicine and traumatic surgery with the following program, Frederick A. Besley, M.D., Waukegan, Ill., Chairman of the Committee on Industrial Medicine and Traumatic Surgery, presiding:

Insurance Company Relationships and Responsibilities in Compensation Insurance, by A. D. Lazenby, M. D., Baltimore, Md.

Treatment of Abdominal Trauma, by Arthur R. Metz, M. D., Chicago, Ill.

The General Management of Injuries, by John J. Moorhead, M.D., New York City.

Hernia in Industry, by John R. Nilsson, M.D., Omaha, Nebr.

Treatment of Old Traumatic Bony Deformities of the Face, by Archibald H. McIndoe, M.B., M.S., London, England.

Prophylactic and Active Treatment of Anaerobic and Micro-aerophilic Infections with Zinc Peroxide, by Frank L. Meleney, M.D., New York City.

Reports of Surveys, by M. N. Newquist, M.D., and Harold Earnheart, M.D., Chicago, Ill.

Association for Advancement

THE Association for the Advancement of Industrial Medicine and Traumatic Surgery held a meeting on September 16, 1936, in the rooms of the Electrical Association, Grand Central Palace, New York City, at 8:30 P.M., at which:

James M. Smith, M. D., read a paper on the Diagnosis and Treatment of Industrial Eye Accidents, which was discussed by Martin Cohen, M.D., Francis D. Gulliver, M.D., Irvin Torok, M.D., and Morris Davidson, M.D., Opthalmologist, Department of Labor, State of New York; and

Joseph Jordon Eller, M.D., read a paper on Industrial Skin Diseases, Causes, Prevention and Treatment (with a lantern slide demonstration), which was discussed by Oscar L. Levin, M.D., Anthony Cipallaro, M.D., and Howard Fox, M.D.

New York Academy

THE Ninth Annual Graduate Fortnight of the New York Academy of Medicine, in October, 1936, is to be given over to a discussion of trauma in occupational diseases and occupational hazards. Among the subjects are the following:—historical background of industrial and occupational diseases; relation of trauma to diabetes, to pulmonary diseases, to cardiovascular diseases, to the nervous system; shock and hemorrhage; accidents and their management; the medical problem in first-aid; fatigue and noise in industry; relation of trauma to malignant tumors, general principles of fracture treatment; hand injuries; toxicology of industrial poisoning; medico-legal aspects of industrial poisoning. In addition, a series of clinics and demonstrations will be held in various New York hospitals.

Industrial Medicine

The Journal of Traumatic Surgery and Occupational Diseases

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VOLUME 5 AUGUST-SEPTEMBER, 1936 NUMBER 9

Injection Treatment of Hernia

DR. FOWLER'S excellent presentation of the current objections to the injection treatment of hernia, which leads off the features of this issue, is the best we have seen. The author is, as he admits, qualified to speak with peculiar authority in the matter; and he has gone to great pains to make his analysis complete and authoritative.

Immediately following his article, we include the recent report of the Council on Pharmacy and Chemistry of the American Medical Association, which indicates that the A.M.A., while not unmindful of the value of the method when rightly used under all proper conditions, is still—as, in the nature of its position, it must be—fearful of wrong use under conditions which are not proper. This is not alone in the non-medical employment of the injection treatment, "by osteopaths and even by laymen", but also in relation to the use of "mixtures of unknown composition" so readily available. The report closes with the injunction:

"Those who inject into the bodies of their patients mixtures of unknown composition, not suitably standarized by any unprejudiced agency, and capable of bringing about definite pathologic changes in living, healthful tissue, may be subjecting themselves to unwarranted risk of legal difficulties should unfortunate results ensue. Those who continue to experiment with the injection method for the treatment of hernia with a view to standardizing this method will do well to use only such mixtures as may be prepared under their own direction in the laboratory of the hospital with which they may be associated, or mixtures prepared by competent pharmacists according to prescriptions prepared by the physician himself."

In the section of the magazine in which we gather articles from various sources which might not, in ordinary course, reach the eyes of a good many of our readers who would hate to miss them, we include several very interesting articles and discussions on hernia in general, and the injection treatment in particular. These, in their entirety, offer a rather good cross-section of the present interest in this subject.

"Cyclopedia of Talent"

WHO'S WHO IN INDUSTRIAL MEDICINE AND TRAUMATIC SURGERY is now completed, and ready for delivery. It is, as such a work should be, only a collection of biographies—more than a thousand—of the important medical men in the field of industrial medicine and surgery. The word "field" is used, because that is what the practice of industrial medicine and surgery is, as distinguished from any specialty. As the preface of the book sets forth, industrial medicine is the superimposition of *all* the specialties of medical practice upon the aggregate, large or small as the employment situation may be, of individuals who are employed; and the doctor-patient relation is a *plus* because it has something added to it as the result of the *fact* of employment.

As a whole, the picture, as one might read the biographies, is that of accomplishment, expressive of inherent ability to accomplish, carefully prepared for the purpose of accomplishing. There is nothing haphazard about a career in industrial medicine.

New Schedule

INDUSTRIAL MEDICINE was established in 1932, as the monthly publication of the field of industrial medicine and traumatic surgery, and with the intent to present a cross-section of the current developments in this field as nearly coincident with the events themselves as the mechanics of journalism would permit. The magazine, therefore, has not adhered to a definite publication date, aiming only at being distributed during the month rather than on or before any certain date of the month.

We find, however, with our greatly increased circulation, and the recognition among advertisers of the value of this magazine as an advertising medium, that there is much to be gained by adopting a fixed schedule.

To this end we announce that beginning with the November number, of this year, the schedule will be the fifth day of each month. Meanwhile we are catching up by combining in this number the issues of August and September, and anticipating the mailing of the October number between the 10th and the 20th of that month.

In response to the reaction among our subscribers that we have outgrown our front cover design, we are also arranging a different, and we believe, a vastly improved front cover.

Industrial Medicine and Surgery

Medical Aspects of Accident Control

THE prevention and control of accidents depend on the cooperative efforts of public authorities, governmental agencies, industrial management and employees, insurance companies, technical societies, safety engineers, physicians and nurses, as well as on the public in general. *Accidents must be combated in the home, in the school, in public places, in travel and in places of work. Administrative, statistical, medical, engineering and educational activities must be covered in any well rounded safety program.

Although much already has been accomplished in safety work, with a marked reduction in the incidence and severity of accidents, much still remains to be done particularly with reference to accidents in the home and in public places and travel. Most of the results during the last two decades in the prevention and control of accidents have been brought about in industries through the development of well organized industrial safety programs. Recent studies made by some of our operating telephone companies in the Bell System indicate that there are three or four times as many accidents to employees while off the job as there are while they are at work, and five or six times as many days lost on account of accidents occurring while away from work as there are while on the job. This emphasizes the need for a far reaching and forward looking program which will concentrate more and more efforts on public and home safety without a let up in well established industrial safety activities. There is little wisdom in devoting time and money to an industral safety service within an office or plant and forgetting the fact that most of the accidents to industrial employees now occur while they are away from their regular occupation. While it is realized that management has no supervision over employees while off duty, the industrial safety program so far as its educational aspects are concerned must "carry over" into the homes and leisure time of employees and must become closely integrated with all accident prevention activities of the community.

Medical Examinations

THE physician's duties and responsibilities in an industrial safety program, while having to do somewhat with various administrative, statistical, inspectional and educational activities, necessarily must be related more specifically to premp'oyment and periodic physical examinations and to emergency medical and surgical examinations and treatment. While early diagnosis through medical examinations is one of the chief factors in the prevention and control of sickness among workers in industry, the inherent value of the preemployment physical examination so far as the prevention of accidents is concerned is in its potential power to make possible the proper placement of workers from the standpoint of accident hazards and prevent prolonged disability and loss of time from work. Good physical condition, as a rule, means more rapid recovery from accidental injuries; poor physical make up is conducive to slow convalescence. Of even more value than the preemployment physical examination, in the prevention and control of occupational diseases and accidents, is the periodic check up. For this reason all employees exposed to special occupational hazards of sickness or injury should have a period medical examination. Just as the newer public health and industrial health focus attention on personal hygiene as of equal value with environmental sanitation, so also a newer public safety and industrial safety must emphasize the make up of the individual worker as contrasted with his environment and mechanical equipment. Accident prevention is becoming recognized as being dependent more and more on medical and psychologic examinations and the proper follow up and placement of employees.

Each industrial organization must decide for itself as to the future development of new phases of preemployment and periodic medical examinations, including routine roentgenographing of the chest and such tests as pertain to the nervous and mental qualities of the prospective employee. Mental hygiene in industry is only on the threshold of development. As interest grows in this important subject, which underlies the whole realm of safety and physical health, more and more industries will be thinking in terms of mental and nervous system examinations in connection with their routine physical appraisals. Although the majority of industries do not include blood and other tests for venereal diseases in their routine preemp'oyment or periodic physical examinations, more and more companies are beginning to recognize the hidden costs in money and disability from accidents and sickness to which these diseases give rise. The prevention and control of syphilis and gonorrhea constitute a health and safety measure which should be of vital concern to the whole community.

First Aid

FIRST AID is the most important adjunct of accident prevention and control; its study and practice promote safety mindedness or safety consciousness. Physicians must assume the leadership in the teaching of first aid, which has been defined as a "special branch of practical medicine and surgery, by a knowledge of which trained persons are enabled to afford skilled assistance in cases of accident and sudden illness, and through which prolonged or permanent disability may be prevented and life often saved."

Without elaborating on its details, which are more or less self evident, the following may be listed as an outline of the fundamental elements of an adequate first aid service:

A. First aid treatment:

1. Immediate reporting of injuries for diagnosis and treatment, together with complete records.
2. Conveniently located first aid treatment facilities.
3. First aid work carried on under trained and experienced personnel.
4. Adequate follow up of each case.

B. First aid instruction:

1. Instruction on principles and practice of emergency first aid, including particularly artificial respiration and the control of bleeding.
2. Printed publicity, group talks, moving and talking pictures and bulletins on subject of first aid.
3. First aid training classes, teams and contests, when practicable and feasible.
4. First aid committees and special campaigns and rallies to stimulate group interest.

The five general principles to be applied as they may be necessary in the practice or teaching of first aid work are:

1. Obtaining a doctor's services.
2. Stopping bleeding.
3. Restoring breathing.
4. Making the patient as comfortable as practicable.
5. Doing no more than is actually needed.

Accident Proneness

FROM various recent studies it is now well established that certain persons have a special liability to be the subjects of accidents just as some individuals are particularly susceptible to diseases. Not only may a small percentage of a working group show a very high percentage of the total sickness disability in the entire group. but it has been found that as little as 10 per cent of a group may be responsible for as much as 75 per cent of the accidents occuring among them. Recent medical research has shown that "accident proneness" may be an innate characteristic of some individuals and a personal phenomenon independent of any question of responsibility, conscious action or blameworthiness. It has been found that those who sustain an undue number of one kind of accident also sustain an undue number of other kinds and that accident proneness is a relatively stable quality obeying definite laws, so that if those persons who have an excessive number of accidents in their first year of employment and exposure were eliminated, the subsequent accident ratio of the group would be considerably diminished. Some have gone so far as to suggest the possible desirability of actually eliminating those who show an undue number of accidents in an initial period of employment in order to reduce the subsequent accident rate of the group and as an important method of accident prevention. While it appears reasonable that a person should be removed from a position in which he is a potential danger to himself and others, indus-

* LEVERETT D. BRISTOL, M.D., DR.P.H., Health Director, American Telephone and Telegraph Company, New York; read before the Section on Preventive and Industrial Medicine and Public Health at the Eighty-Seventh Annual Session of the American Medical Association, Kansas City, Mo., May 15, 1936, and published in *J.A.M.A.*, August 29, 1936.

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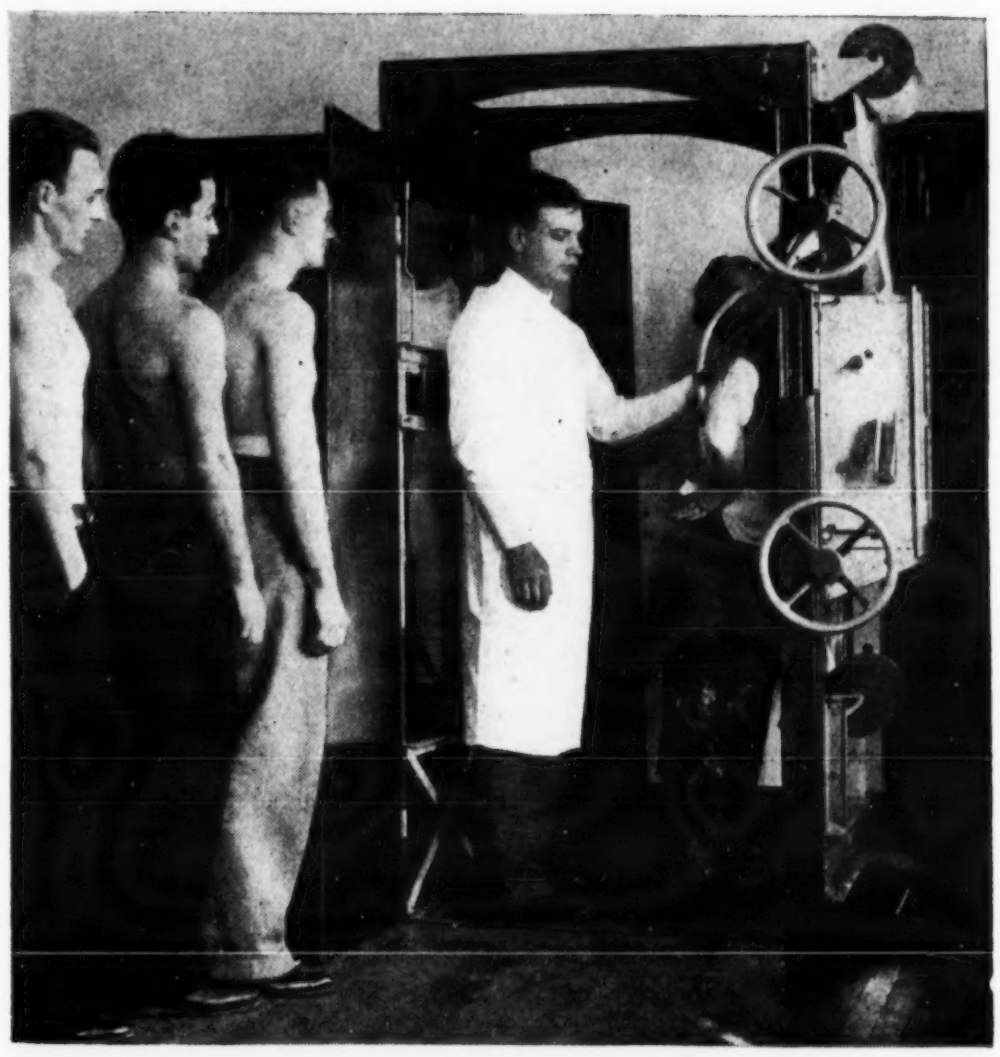

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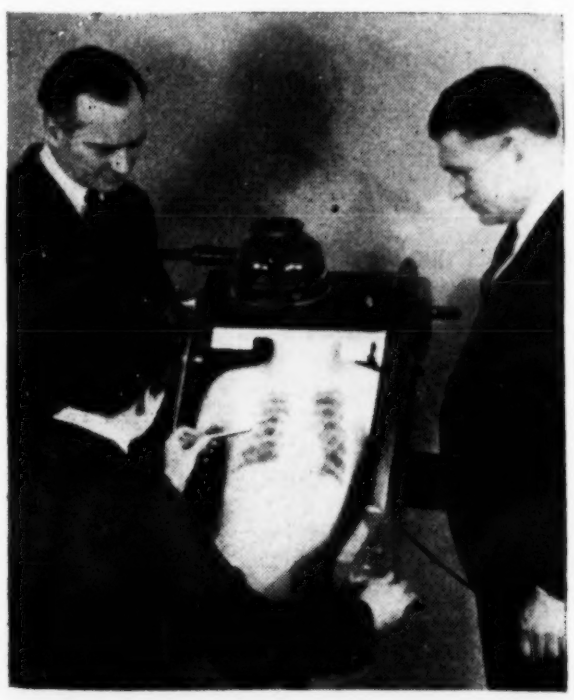

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tries realizing their obligations to society in general undoubtedly will be slow to penalize an individual because, through no fault of his own, his mental and physical qualities are not equivalent to those of his fellow employees. In this connection the chief objective should be to see that these accident-prone individuals, who may make up as much as one fourth of a working group, are identified through medical and other examinations and are assigned to work in which the severity and danger of their potential accidents are reduced to a minimum. Moreover, the accident prone generally are the least healthy, indicating that the prevention of accidents may depend to a considerable degree on the prevention of sickness and emphasizing further the need for greater attention to the entire health program. Individual accident susceptibility may be based on such factors as (1) the effectiveness of the visual apparatus, (2) reaction times, (3) reduced energy, (4) the intensive and extensive distribution of attention, and (5) certain kinesthetic impressions or sensations and nervous reflexes making possible more or less automatic performance. Newer developments in regard to the relation of some of these factors to the incidence of accidents should be mentioned briefly in passing.

Vision

ASIDE from various pathologic conditions there are two factors which in the past have served to determine whether or not the eyes of an individual are functioning properly; these are refraction and muscle balance. Recent studies by Ames[1] and his co-workers indicate that there is a third newly discovered factor of importance; that is, the relative size and shape of the ocular images of the two eyes, a difference in which constitutes a condition known as aniseikonia. In such a condition, depth perception or stereoscopic vision is seriously disturbed and it is conceivable that such impaired vision may be a very important element in the causation of accidents. To insure perfect single binocular vision it is imperative to detect, measure and correct aniseikonia, and this may come to be one of the newer requirements in the control of accidents from the standpoint of vision.

Reduced Energy

ACCIDENT proneness, or individual accident susceptibility, at times has been explained on the basis of physical, mental or nervous fatigue, such fatigue usually being interpreted as reduced or impaired energy due to some particular type of work, labor or exertion. Recent studies by Haggard and Greenberg[2] seem to indicate that drops in efficiency and productivity may not necessarily be related to actual fatigue and weariness relieved by rest, but that many cases of so-called fatigue are nothing more than reduced energy due to the need for and relieved by food.

A recent study of personal injury accidents made by one of our companies shows that the great majority of accidents of this nature occur between 10 and 12 o'clock in the forenoon and 3 and 5 o'clock in the afternoon. As the work involved and the rest periods available preclude the possibility of so-called industrial fatigue, it is conceivable that these accidents were related to reduced energy, which may have taken place with the approach of meal time and the need for food. While preliminary experimental studies point to the possible value of extra feedings in the midforenoon and midafternoon, to prevent loss in efficiency, effort or production and possibly to reduce accident susceptibility, it would seem wise before attempting the expense and trouble of providing extra feedings as a routine to be sure that the industrial worker, the school child and the housewife have the necessary knowledge as to their average food needs for a day and that they become thoroughly acquainted with the fact that breakfast should be something more than a cup of coffee and that a sandwich is not a complete lunch; all of which, in the interest of safety as well as of health, argues for more instruction on diet and nutrition not only for household members but for school children and industrial workers. What first may be necessary is not more but better balanced meals, and this is largely a matter of education. Before advocating an increase in the number of meals per day, as some have done, it would be wise to make certain that the three regular meals are what they should be in quality and quantity.

Nervous Reflexes

"THE car went out of control" has been the explanation of many automobile accidents in the past. According to the recent suggestion of Henderson[3] it often in reality is the motorist "who goes out of control," thus emphasizing further the fact that the newer public safety must concentrate more and more on the human element, including individual nervous reflexes and reactions. The so-called self-righting reflex caused by a sudden upset of equilibrium in an emergency and the more or less unconscious "extensor thrust" of the legs and feet are instinctive reactions which may result in a sudden pressure on the accelerator pedal of an automobile, this causing a serious accident although the car itself and its motor, brakes and steering gear are in perfectly good order. All of which is a further indication that machines and equipment should be fashioned for the human individual and his safety, based on a complete knowledge of his physical requirements and nervous reflexes, which can be determined only by suitable medical examinations. Compulsory periodic testing of automobiles already is in effect in some states; required periodic medical examinations of all licensed vehicle operators should be the next step in highway safety.

Home and School Safety

IT IS becoming more and more apparent that safety in the home should be the central objective and influence from which radiates safety in all other walks of life. To this end, instruction in safety practice is of paramount importance in the home. In this great work, no individuals are in a more strategic position to accomplish results through practical teaching than are the physician and the nurse.

Safety in industry, in the school, in public places and in travel has been built largely on the combined foundations of enforced regulations and education. In the home, on the other hand, most of our results in reducing accidents must be accomplished through education alone. Home accidents, as a rule, are due to ignorance, indifference or inadequate equipment. Both the human element and the home environment are the factors to be considered. To a large degree, the problem is one of education of individuals, preferably in family and home groups. Ignorance and indifference must be dispelled and proper methods of using home facilities and equipment taught.

Health and welfare problems more and more are being solved with the family and home as the unit to be considered. So also the problems of safety must be approached through the home, not only for the benefit of the home itself, but also in the interest of furthering all phases of human safety. The family doctor in carrying on his functions as a teacher of health and a healer of disease should grasp every opportunity to teach the facts with reference to accident causation and prevention to the children and adults with whom he comes in contact. Moreover, medical examiations of individuals in family and school groups should be of as much value as exminations of industrial workers in bringing to light the accident prone and in serving as a background for safety education. Such medical examinations in industry, in the school and in the home will contribute also to the control and prevention of accidents in public places and in travel.

Training of Physicians

FINALLY, it may be stated that better facilities should be developed in our medical schools for teaching the medical aspects of accident prevention and of safety; and adequate graduate instruction should be made more generally available for those planning to specialize in these fields. Prospective doctors, many of whom eventually will be called on to render service particularly in the field of industrial accidents and safety, should be taught the principles of court procedure and testimony, the many aspects of liability and compensation, and the fundamentals of personnel administration and industrial relations. The medical curriculum is in need of various modern amendments to permit it to keep pace with the growing demands made on the intelligence and ability of the present-day physician. In no field are these demands greater than in that of preventive and industrial medicine, public health and safety.

Abstract of Discussion:

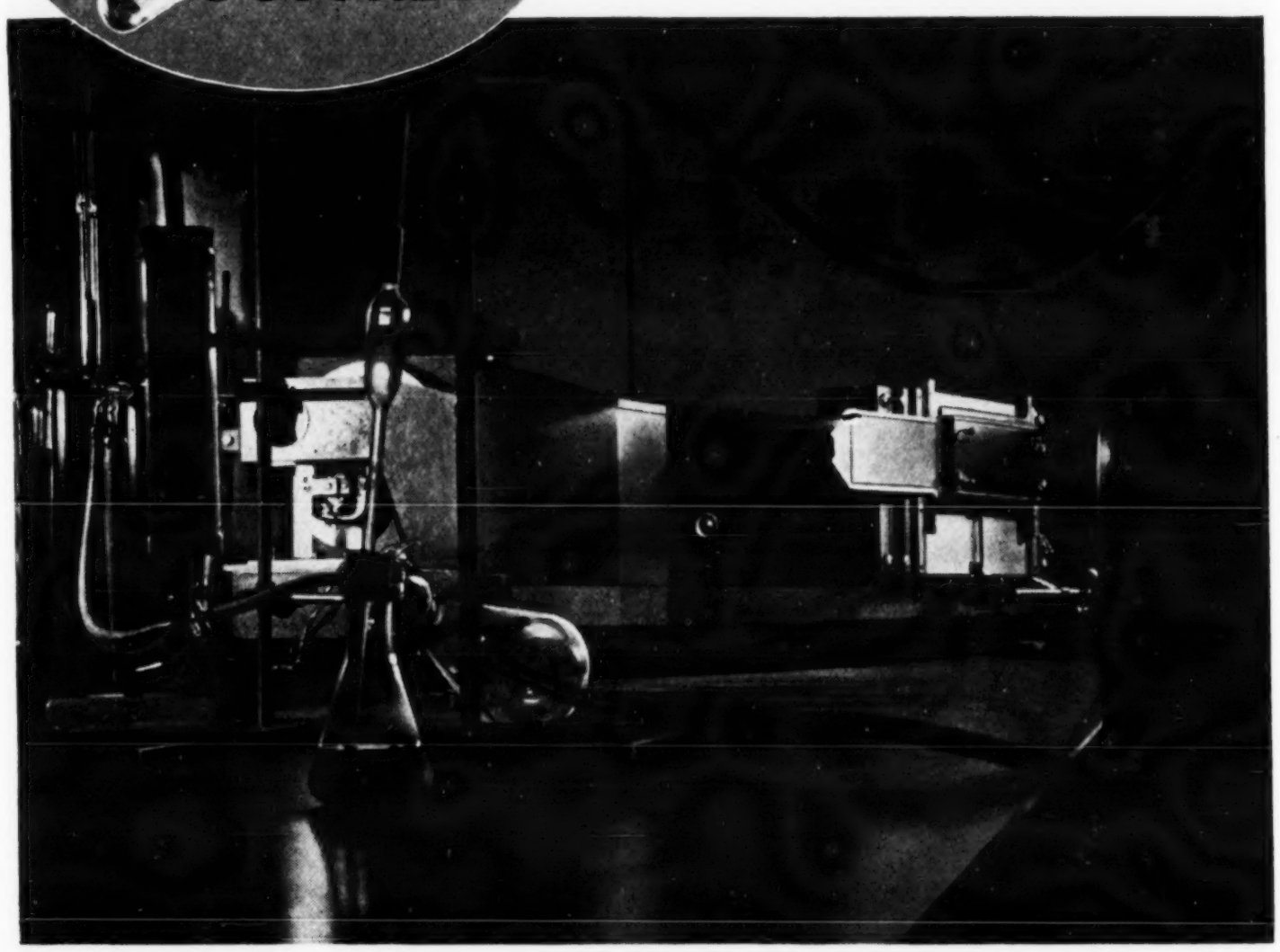
DR. JOHN H. OGILVIE, Kansas City, Mo.: I was interested to see that Dr. Bristol's problems in the prevention of accidents have been

1. AMES, ADELBERT, JR.: Aniseikonia: A Factor in the Functioning of Vision. *Am. J. Ophth.* 18:1014 (Nov.) 1935.

2. HAGGARD, H. W., and GREENBERG, L. A.: Diet and Physical Efficiency, New Haven, Yale University Press, 1935.

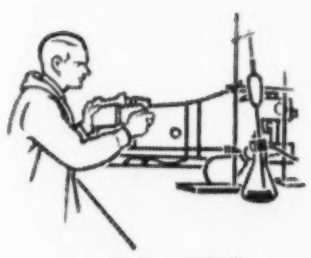
3. HENDERSON, YANDELL: How Cars Go Out of Control: Analysis of the Driver's Reflexes, *Science*, 82:603 (Dec. 27) 1935.

Curity SUTURES and the Spectroscope



SCIENCE is AUTHORITY

FOR CURITY QUALITY



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very similar to mine in a work that is entirely dissimilar to his; that is, the same factors are present in Dr. Bristol's work and in mine. I should like to show some results of this individual whom he calls the accident-prone individual. He is a very potent source of expense in industry. Years ago I began a study to see what I could do to reduce my medical costs. Some interesting things were found: First, the medcial cost and the proportion of serious accidents were in direct proportion to the number of minor accidents; second, that 16% of individuals were contributing about 66% of the accidents; so I began a study of this small group of individuals. After suggestions from medical men and safety engineers, and all the safety devices we could devise, I found that the proportion of accidents was still too high. I began a further study to determine the cause of these accidents and came to the conclusion to which Dr. Bristol has come, that there is an individual who is accident prone. I find that this individual falls into three classes: (1) the individual who is hindered by some major or minor physical defect, (2) the individual who is in a job that is not suitable to him, and (3) the individual who is accident prone simply because he cannot be educated. He is the person who is involved in a great many of the major accidents. He is analogous to the man who walks across the street through a stream of traffic reading his newspaper. In the first group I found that by repeated examinations during the course of emp'oyment, that individual could be eliminated; that is, by sending him to his local doctor and making suggestions to him, I could correct him. In the second group by being fitted into a job which was suitable for him, the individual could be corrected. In the third group, individuals who could not be educated in spite of an educational campaign and safety hazards education, the only thing that had any effect was disciplinary measures. Only 30% of the accidents to which I have been referring that occurred ten years ago are now happening. I am glad that Dr. Bristol has drawn attention to these things that are associated with this accident prone individual.

DR. LOUIS J. HIRSCHMAN, Detroit: I offer an apology for speaking at this meeting because I practice clinical medicine; however, I am a member of the Michigan State Council of Health and I am interested in public health as well as in the individual. I also come from a city where the most dangerous weapon on which man has his hands—the automobile—is manufactured. It is said that automobile accidents are responsible for more lives lost than any other one class of hazards. Much is heard about the drastic action of courts on careless drivers; but it seems to me that a leaf should be taken from the book of the Department of Commerce in connection with the rigid examinations of airplanes as well as of pilots. There should be, in a lesser degree, some definite method of periodic examination of automobiles with regard to brake control, as well as an examination of the weakest part of the automobile; that is, "the nut at the wheel." There should be some way of examining him before he receives his driver's license, an examination by his family physician, the same as in the case of the locomotive engineer, the bus driver and the taxi driver. If definite action could be taken by which municipalities would insist on more than a cursory examination of drivers, and the physically and mentally unfit eliminated, a great deal would be done toward improving the medical aspect of accident control.

DR. STANLEY H. OSBORN, Hartford, Conn.: I think that the last speaker has landed in the middle of an interesting problem. I should like to make a motion that the incoming chairman of the section appoint a committee on accident control and safety to study the medical aspects of accident control and make recommendations to the next annual meeting.

The Industrial Nurse

THE subject for consideration is: What are the qualifications of a good industrial nurse? *Like the practice of medicine, the profession of nursing has become more and more specialized. In addition to the standard course of instruction given in all good schools of nursing, a nurse desiring to qualify for specialized work is required to take additional training or to acquire, through experience, other knowledge which will better qualify her for the position to be filled.

There are certain fundamental requirements for a successful nurse: for example, poise, personality, intelligence, loyalty, honesty, and physical endurance. These requirements she must measure up to and without them she should not be graduated from a school of nursing. In addition, there are special qualifications which are necessary if a nurse is preparing to enter upon an industrial career.

The industrial field also has a number of branches, which require some variation in the selection and training of those who do industrial nursing. While there may be other branches than those included in the following classifications, I believe that, in general, we may consider them under these headings:

1. The office industrial nurse.
2. The plant nurse.
3. The plant nurse, in large and more or less hazardous manufacturing activities.
4. The visiting industrial nurse, in corporations having extensive social service activities.

There are some important qualifications which are sought for in any type of industrial nursing, and which are necessary for a smooth-running, well coordinated program. My ideas about these qualifications are, in all probability, somewhat arbitrary. However, they have been developed over a period of many years of experience in training in experienced personnel or selecting previously trained and experienced workers and adapting them to the needs of this work.

An industrial nurse should be mature in mind if not actually in years. She should be physically sound and able to endure hardships under trying circumstances. She should have a pleasant, and at the same time a firm personality. Her social relationships and her professional relationships should be well separated by a line of significant demarcation. However, she should strive to deserve the respect and regard of those with whom she mingles daily in the course of her professional duties.

The nurse should either know or be capable of acquiring a knowledge of the complex and intricate aspects of the industry in which she is an integral part of the personnel. She should familiarize herself with the entire personnel from the general manager to the watchman and in so doing develop that rare quality of being able to assist in the promotion of friendly relations between the employee and the employer, a consideration of paramount and vital importance in all corporate organizations of today.

The industrial nurse should also learn about the attitude of labor toward working conditions and should be able to analyze and evaluate circumstances and conditions as they develop or occur in the course of her day-to-day duties. She should be on the alert to observe what is going on about her but talk little or not at all unless she or her medical department is directly concerned.

She should be loyal to the workers and also to her employers; and from her point of vantage in the organization she should be able to quietly yet impartially see and assist in the handling of the many daily problems—problems not always having to do with bandages and antiseptics.

The scope and value of her service will be broadened if she knows how to use the typewriter and can take stenographic notes. A good knowledge of anatomy and the proper use of medical terminology will greatly add to her efficiency and will promote smoothness and effectiveness in her day-to-day production on the job.

While we do not expect the industrial nurse to be a first aid surgeon, we do expect her to have an understanding of the principles and practices of modern surgery. She should know thoroughly the ordinary standard American Red Cross first aid practices. Although her training in surgical technique should, of necessity, extend far beyond the scope of Red Cross practices, yet as a beginning they are invaluable. Proper surgical technique is of paramount importance, but it cannot be carried out without a systematic orderliness and neatness—so quick to be noticed and approved by all who come in contact with the nurse.

Gentleness and sympathetic consideration are two of the qualities expected of all nurses at all times, but I especially refer to them here because the average worker with whom the nurse comes in contact has a feeling—and I know not why—that we in the health service are always in a hurry, that this is characteristic of our daily routine, and that we are going to be rough and hurried in our treatment of the patient. The indus-

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* PHILIP STEPHENS, M.D., Medical Director, Southern California Telephone Company, Los Angeles, California; presented before the N.O.P.H.N. Round Table for Industrial Nurses, Biennial Convention, Los Angeles, California, June 24, 1936; published in *Public Health Nursing*, September, 1936.

trial nurse should have the faculty of demonstrating by quietly spoken words, and then by action, that she can and will be gentle and in so doing dispel this prejudice, thereby creating confidence in the health service.

Most of the personal qualities which I have mentioned might be crystalized into the one word, tactfulness—but this word as applied to an industrial nurse needs much amplification.

The keeping of accurate records is highly important. The accurate, pertinent history of events written down at the time of an accident go far toward the prevention of controversial litigation which may arise in the course of the subsequent care and disposal of an injury case, and such a record materially assists the doctor in his work and results. Many pertinent incidents are related at the time of the first examination in an accident case which, if promptly and accurately recorded, clear up problems which develop later due to the faulty memory of the patient. I do not mean that the nurse should be entirely responsible for the promptness and accuracy of injury reports, but she should assume a large part of it.

In considering the qualifications of the industrial nurse in the office of an average business organization, we might say that she should possess all of the qualities mentioned in the foregoing, beside having a special aptitude for clerical duties. She can be of great assistance also if she acquires a knowledge of laboratory technique. The average office doing some minor surgery will usually be equipped with a small modern x-ray laboratory; and the nurse who is able to make x-ray exposures of the extremities and develop them will be of particular assistance to the surgeon and will thus relieve him of the necessity of sending out these semi-emergency injury cases to x-ray laboratories or nearby hospitals.

The nurse expected to function in a plant where hazardous work is performed, and where major injuries occur, must be especially fitted to cope with disasters. Usually she is not alone, but acts as an assistant to a plant physician who is continuously on duty or at least within ready call. In these positions where there is the necessity for the nurse to assume considerable responsibility in giving the immediate first aid so often necessary in major accidents, she should be alert, capable, and of forceful character. She should not only be able to handle her surgical and first aid problems, but also have a sufficiently dominating personality so that she can handle excitable, and very often, hysterical fellow workmen, who are inclined to get out of hand at these times of emotional stress and strain.

The industrial nurse who also acts as a personnel assistant must have special training for this work, as there are many sociological problems continually presenting themselves. In addition to a sound understanding of human relationships, it is desirable that she should be especially trained along social service lines or at least should understand the fundamentals of that service, which will help her with the many problems arising among the working personnel.

In summarizing our remarks, we might say that a good industrial nurse should be fundamentally a good nurse with a sense of obligation to her fellow man. In addition to this, she should be mature mentally, strong physically, broad and tolerant in her views of life, dignified and yet friendly, loyal to her profession, and willing, if need be, to improve and perfect herself in various other closely related branches in order to adapt herself to the changing requirements of specialized work.

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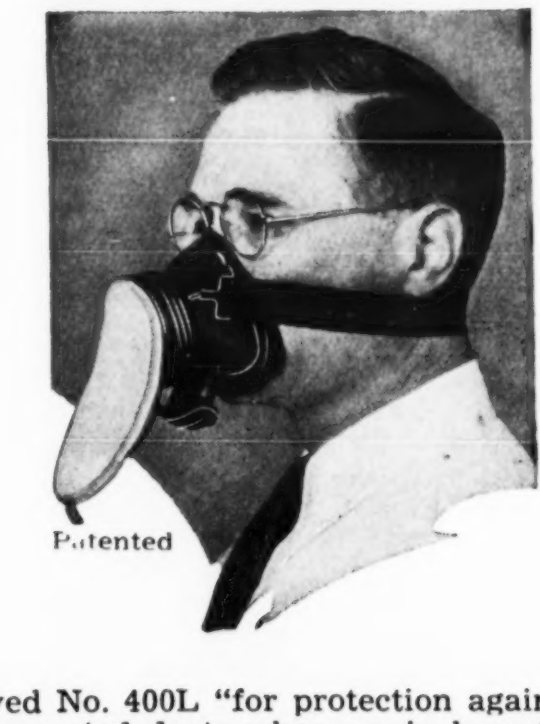

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ed and well prepared nurses, I feel that our schools of nursing should take cognizance of this need and should meet it either by incorporating the teaching of the special requirements in their present nursing courses or by inaugurating special courses to cover the much needed additional requirements for successful industrial nursing.

Essentials in the Diagnosis of Silicosis and Tuberculosis

PNEUMOCONIOSIS should be defined as a condition of the lungs resulting from the prolonged inhalation of dust.* Only when this condition is due to a type of dust capable of producing serious and progressive changes in the lungs can a pneumoconiosis be considered as a disease. At the present time only two kinds of dust are generally recognized as possessing such irritating properties. Silica, commonly found in nature as quartz, is a notorious offender, and asbestos, a silicate of magnesium, is as bad if not worse. Some of the other silicates may possibly cause trouble, but assumption of harmfulness without proof is quite unwarranted. As industrial surveys become more frequent, we shall be in a better position to evaluate the hazards involved.

A diognosis of either silicosis or asbestosis depends primarily upon a reliable history of adequate exposure to silica or asbestos dust, and upon the demonstration of characteristic changes in an x-ray film of the chest. Physical findings are important in the detection of complicating infections and in estimating disability if it is present. The roentgenogram is often sufficiently characteristic to make one suspect either of these two conditions, but unless the suspicion can be confirmed by a history of exposure to either dust, a positive diagnosis must not be made.

What constitutes an "adequate exposure?" A man must be exposed day after day for a period of years to relatively high concentrations of dust. In the usual industrial concentrations of silica dust it takes from five to 20 years to produce silicosis; and from seven to 11 years in asbestos to produce asbestosis. In rare cases, where the dust concentration is excessive, the time may be shortened to two years. Not only time but dust concentration is an essential factor in exposure. The determination of dust concentration is a technical engineering problem involving the use of special apparatus, but there are certain operations where it is safe to assume that dust concentrations are high unless somewhat elaborate precautions are being taken to prevent the formation of dust or its inhalation by the men. Mining in hard rock, particularly where dry drilling is involved, cutting and polishing granite, grinding with sandstone wheels, and sand pulverizing are notoriously hazardous occupations. There are other industries such as foundries, potteries, and paint-manufacturing plants that use silica and may have silica hazards in certain departments.

The fabrication of asbestos apparently involves considerable danger, particularly when it is handled in a dry state. It is used in the manufacture of fireproof textiles like curtains, brake linings, steam packings, and conveyor belts. As building material it is made into shingles, wallboard, and insulation, and it has various uses in electrical work.

While a history of employment in any of these industries that use either silica or asbestos should put one on his guard to suspect pulmonary disease, he should enquire further to make certain that the particular occupation actually involved an exposure to dust. A patient might say, for example, that he worked in a pottery, but on closer questioning disclose that he was actually engaged in decorating the finished product. On the other hand, a man whose x-ray film showed an apparent silicosis gave his occupation as an axe manufacturer. Subsequent questioning disclosed that his office was a small room loosely partitioned from the one in which axes were ground. Finally, the occupational history must include not only the individual's present or last occupation but a chronological record of the time spent in every job since leaving school. Silicosis is a chronic disease that may last for years; persons who become incapacitated frequently find light work. Record of employment as elevator man or night watchman would suggest nothing, but a history of dry drilling 10 or 15 years previously often presents the key to a case of chronic pulmonary disease.

To summarize: A proper occupational history involves a complete record of the individual's industrial employment. It should include the duration of each job, with details as to the types of work that may involve exposure to dust. Where doubt exists, the kind of dust should be determined by consultation with the employer. In many industries, information is becoming available as to the actual concentration of the dust in different parts of the plant. This may or may not be available to the doctor who sees a patient in private practice.

The other important basis of the diagnosis of silicosis or asbestosis is a characteristic roentgenogram of the chest. The shadows seen on the film are cast by pathological lesions in the lungs, and hence the latter are of fundamental importance. The changes produced by uncombined silica are very different from those caused by asbestos dust, and hence they must be described separately.

Pathology of Silicosis

WHILE there are certain preliminary changes produced in and about the lymphatic system of the lung by the inhalation of silica, the disease, clinical silicosis, does not begin until nodules of fibrous tissue develop in its parenchyma or functional portions. These nodules are quites characteristic. In the absence of infection, they are uniform in size and distribution. They measure from 1 mm. to 5 mm. or at most 6 mm. in diameter. They have sharply defined borders and are surrounded by normal lung. They are composed of masses of very dense, hyaline, fibrous tissue in whose centre is usually a collection of silica particles.

In certain individuals the nodules are more numerous in localized parts of the lung, and they tend to become confluent. It seems probable that such reaction occurs because the particular portion of the lung had been damaged. Perhaps a pneumonia did not completely resolve and left a small amount of scar tissue in which the inhaled silica particles were entrapped and consequently produced an unusual amount of reaction in the local area. Perhaps the scar tissue of a healed tuberculous focus persisted to have the same effect. While due in most cases to infection, the process has healed and the mechanical effects of tissue distortion are responsible for the effect. These two conditions, the generalized discrete nodulation and the generalized nodulation plus localized conglomerate foci, constitute the changes found in simple or uncomplicated silicosis.

Within limits silicosis is a progressive disease even after exposure has ceased. When more particles have been inhaled than can be segregated in the lymphatic system, they accumulate in the walls of the air spaces and are not eliminated to an appreciable extent. Remaining in the lung these particles continue to excite the formation of scar tissue. Nodules that have already started to form increase in size but probably not a great many new ones develop. A generalized nodulation that is either undetectable or is barely visible in a roentgenogram taken when a man leaves a dusty trade usually progresses to well marked proportions in subsequent years. Since progression is practically inevitable, it seems to make little difference whether a person in this condition remains in his occupation or not. Continued exposure simply means more nodulation.

Another characteristic feature of this disease is its tendency to increase the susceptibility to tuberculosis and possibly other pulmonary infections. Tuberculosis may arise from a reactivation of latent foci already present in the lung or it may develop from a fresh implantation of bacilli from external sources. A variety of pathological pictures is produced that are either superimposed upon the generalized nodulation of silicosis or are so intimately combined with the silicotic reaction that it is impossible to separate the two processes. The following manifestations are generally recognized:

1. The primary complex of childhood tuberculosis consisting of one or more subpleural tubercles with associated foci in the lymph nodes in the meriastinum. These are usually healed and partially calcified by the time a person enters a dusty occupation so that they play little part in the development of subsequent disease. They can become reactivated by silica so that subjection of children to such employment would entail grave responsibility.

2. The more or less completely healed foci of adult type tuberculosis that occur most commonly in the apex of the lung. These frequently enmesh and retain more dust than other portions of the lungs so that

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* Dr. LeRoy U. Gardner, at the 1935 Convention of the International Association of Industrial Accident Boards and Commissions, published in "Discussion of Industrial Accidents and Diseases," Bull. No. 4, U. S. Department of Labor, Division of Labor Standards.

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the silicotic nodulation becomes unduly prominent in their immediate vicinity. They may surround a small cavity in which living tubercle bacilli survive for a long time. The silica accumulated in the walls of such cavities tends to penetrate into their degenerated centers. The bacilli then take on new capacity for growth and spread of the disease ultimately follows. As a consequence it is not good practice to employ young persons with even an apparently healed tuberculous focus in a silica industry. As they grow older the chances of viable bacilli being present in the quiescent focus progressively diminish and the risk of reactivation decreases.

3. Bronchogenic spreads. — The most common pathway for spread of chronic tuberculosis in the nonsilicotic individual is the bronchi. Such extensions are manifested by the formation of clusters of tubercles in the terminal branches of some of the tubes. The tuberculous lesions may develop as exudative or pneumonic foci or they may be proliferative, i. e., consisting of tuberculous granulation tissue in various stages of organization. The difference is probably due to the local dosage and to the degree of tissue sensitiveness that happens to obtain at the time of dissemination. Such extensions have their origin in older cavities that are usually situated in the upper part of the lungs. The same type of disease may occur in the silicotic lung but the acute penumonic form is quite rare.

4. Miliary tuberculosis in which great numbers of minute tubercles develop as a result of accidental blood-stream invasion occasionally occurs as a terminal event in the silicotic subject. Many of the tubercles localize in normal portions of the lungs, but in addition many of the silicotic nodules become infected and their centers caseate. About their periphery there may be a rim of tuberculous reaction.

5. Perinodular tuberculosis.—The centers of many of the silicotic nodules are caseous and about their periphery is a zone of inflammatory tuberculous exudate. There is no generalized tuberculosis but an older localized focus is found at some point in the lung. Whether this condition usually follows a miliary tuberculosis as just described, or whether it can also be produced by bronchogenic dissemination is not altogether clear. It can be produced experimentally by intravenous injection of tubercle bacilli into silicotic rabbits, and it is occasionally observed in roentgenograms of living subjects. By the time such persons die and come to autopsy their lesions have progressed to an advanced stage so that they are difficult to analyse. In the only case from which the writer has had opportunity to study tissues, the lesions seemed to have developed from a bronchogenic spread.

6. Silico-tuberculosis.—This consists of a massive, focalized area of chronic disease composed of an intimate combination of silicosis and tuberculosis. The dust reaction is composed of nodular and diffuse hyaline fibrosis in and about a matrix of tuberculous granulation tissue. Caseation is minimal, and when it occurs calcification often follows rather quickly. Cavities never form in many cases; in others they are a rather late manifestation. Chronic, adhesive, fibrous pleurisy is a regular complication. By a slow peripheral extension the reaction may come to involve wide areas in the lung. Such lesions occur in the upper or midportion of the lungs; not infrequently they are bilaterally symmetrical. Elsewhere throughout the organ one usually finds discrete nodules of simple silicosis, although in many cases they are not numerous. Apparently most of the inhaled dust is entrapped in the area of infection. Such disease may result from reactivation of a latent focus of preexisting tuberculosis. This seems particularly likely when the focus occurs in the upper lung; if the focus occurs in some other part it possibly comes from exogenous sources. Dyspnoea is a prominent symptom but manifestations of intoxication (night sweats, tachycardia, fever, loss of weight, etc.) and the presence of tubercle bacilli in the sputum often fail to appear until the disease has progressed for years.

Roentgenological Appearances

THE character of the shadows cast by these various pathological changes can now be described. The terminology and description used to designate the different appearances is that suggested by a committee whose report was published in the United States Public Health Reports, August 2, 1935.

1. Nodulation of simple silicosis is characterized by "discrete shadows not exceeding 6 mm. in diameter, tending to uniformity in size, density, and bilateral distribution with well defined borders surrounded by apparently normal lung shadow. The outer and lower lung fields characteristically show fewer nodules."

2. Conglomerate disease of simple silicosis is characterized by "conglomerate shadows that appear to result from a combination of or consolidation of nodulation usually with associated emphysematous manifestations" (localized increased transparency and contrasting intensification of the trunk shadows).

When infection is added to the picture, any of the following may occur upon a background of generalized discrete nodulation:

3. The primary complex of childhood tuberculosis with its isolated calcified, subpleural tubercle or tubercles and associated calcification in the lymph nodes at the root of the lung.

4. Localized healed tuberculous foci either apical or basal in distribution characterized by "localized discrete densities with or without string-like shadows."

5. Clustered foci of aspiration tubercles characterized by "mottling." "The shadows vary in size, have ill-defined borders, and lack uniformity in density and distribution."

6. Miliary tuberculosis superimposed upon extensive generalized discrete nodulation may be very difficult to identify in a roentgenogram. The diagnosis is more often made at the autopsy table. As previously mentioned, the next group—

7. Perinodular tuberculosis reaction or soft nodulation may follow either bronchogenic or blood-stream infection of the silicotic nodule. Roentgenologically "the nodular shadows described under simple silicosis (1) have now assumed fuzzy borders with or without irregularities in distribution." Associated mottling (5) may also occur in localized areas.

8. Chronic silico-tuberculosis is manifested my "massive shadows of homogeneous density not of pleural origin symmetrically or asymmetrically distributed."

The last type of shadow produced by the lesion of chronic silico-tuberculosis is difficult to differentiate from the conglomerate shadow of simple silicosis (2) where the active phases of the infection have entirely disappeared. Serial films, clinical and bacteriological examinations, overexposure to penetrate the dense homogenous mass in the hope of detecting areas of cavitation or calcification, may all be necessary to make the distinction. One observation of such cases is not sufficient to establish a diagnosis.

The changes mentioned comprehend the various phases of the disease, silicosis. Previous to the development of characteristic nodulation in the pulmonary parenchyma, inhaled silica dust accumulates in the lymphatic system of the lung and causes a relatively slight amount of reaction in the surrounding connective tissues and some enlargement of the lymph nodes and nodules. Since lymphatics run through the walls of blood vessels and bronchi, these structures appear slightly thickened. In a roentgenogram such changes are manifested as an accentuation of the linear markings in the lungs. However, this reaction is not specific; it may be produced by many types of dust, by chronic infections, and by cardio-vascular disease. Even though an individual has worked in a silica industry a simple increase in the linear markings of his lung fields does not constitute a basis for a diagnosis of silicosis. To have the disease there must be nodules in the parenchyma of the lung. The linear exaggeration in the film might be due to slica, and it might be due to other forms of irritation. It cannot be emphasized too often that without parenchymatous nodules a diagnosis of silicosis is unwarranted.

Pathology of Asbestosis

INHALED asbestos particles are not collected in masses within the lung tissue. They come to rest in the walls of the terminal bronchioles and excite the formation of scar tissue in this location. The result is a cuff of fibrosis in and around these small tubes. As this contracts it narrows the tube and finally prevents the air from penetrating into portions of the lung supplied by the bronchiole in question. This portion collapses and then undergoes fibrosis as a result. Not all of the bronchioles are involved; some remain patent and their peripheral air spaces remain distended. In cutting across an area of advanced asbestosis one finds an extensive obliterating fibrosis including isolated foci of apparently normal air spaces. Such a picture is difficult to analyze, but the evolution of the process has been observed in experimental animals. A character-

istic feature of asbestosis is the occurrence of large numbers of the so-called "asbestosis bodies" in the scar tissue. They are golden yellow in color, due to their high content of iron. They are formed by the development of a haustrated deposit upon the surface of the mineral fiber. A great variety of bizarre forms can be found.

Upon cross-examination the uncomplicated asbestosis lung usually shows bands of gray fibrous tissue situated beneath the pleura with prolongations into the deeper portions. The interlobar septa are thick, and careful examination reveals deposits of slightly pigmented fibrous tissue along terminal bronchi that happen to be cut longitudinally. Cross section of these structures appear as ill-defined areas of fibrosis. Nodular reaction is absent. The lymph nodes at the root of the lung are not appreciably involved.

The relationship of asbestosis to infection is not clearly established. In the cases that have died and come to autopsy, tuberculosis has been found in many instances though it is apparently not as frequent as in silicosis. Others have shown a complicating broncho-pneumonia while still others have developed cardiac complications. Surveys of asbestos workers still alive in this country have not disclosed significant amounts of complicating tuberculosis. There is urgent need for a study of more cases.

The roentgenographic appearance of asbestosis is characteristic but not so striking as that of silicosis. The normal markings in the middle and lower portions of the lung are partially obscured by a fine diffuse haze, giving the film a ground glass appearance. Still later in the course of the disease the appearance is that of very fine stippling that obliterates most of the normal markings. The pleural shadow is definitely thickened. In some of the advanced cases the heart is enlarged, and radiating from it into the lung fields is a series of heavy fibrous bands. The picture has been referred to as the "porcupine heart." The presence of air-containing spaces throughout the areas of fibrosis apparently allows the transmission of a sufficient number of x-rays so that the shadow of the fibrous tissue is not extremely dense but assumes the characteristic ground-glass appearance.

Manifestations of tuberculosis and other infections in asbestosis are similar to those in silicosis. The occurrence of localized dense homogenous shadows and mottling suggests infection.

The clinical aspects of silicosis and asbestosis cannot be discussed in detail in this paper. However, a few points deserve mention. In simple silicosis with no areas of conglomeration there are practically no symptoms. Many cases are discovered in routine surveys that have developed to an apparently alarming degree without the knowledge of the subject. They show no evidence of dyspnoea nor incapacity for work. However, individuals with conglomerate foci do complain and often suffer severe limitations on physical effort. Dyspnoea is the cardinal symptom but is associated with localized fibrosis. It may be the emphysema that is associated with such reaction which is responsible. The effect of other factors like loss of elasticity of the lung and interference with the pulmonary circulation need further study. The purely mechanical effect of replacement of functional lung by areas of fibrosis is inadequate to explain the clinical picture.

To depend upon the clinical picture of intoxication or the presence of tubercle bacilli in the sputum for a diagnosis of tuberculosis in a silicotic subject is a mistake. The complication is usually well advanced before these manifestations develop. Roentgenographic evidence of infection is often present for years before a diagnosis can be substantiated by these methods.

The question as to the frequency of cardiac complications in silicosis is frequently raised. While many observers stoutly maintain that disease of the right heart is unduly common in silicotic subjects, it is debatable whether its frequency it greater than would be expected at the age when silicosis is most often fully developed (40 to 60). Probab y it is rarely produced by simple discrete nodulation. In cases of extensive conglomerate disease or of widespread silico-tuberculosis so much of the pulmonary capillary bed may be destroyed that embarrassment of the lesser circulation is more readily conceivable.

The severity of the clinical picture in asbestosis seems out of all proportion to the apparently slight diffuse haziness seen in a roentgenogram of the chest. However, the widespread fibrosis seems to destroy a sufficient

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number of alveolar walls to seriously interfere with pulmonary function. Interchange of gases can no longer take place and the contracting scar tissues compress and obliterate the capillary blood vessels. Marked dyspnoea and secondary right heart disease are the usual results.

Recommendations for the treatment of cases of silicosis vary with the experience of different students of the disease. It is the writer's belief that no treatment or change of occupation is necessary in the case of the man whose roentgenogram, perhaps taken in a routine industrial survey, shows only widespread discrete nodulation without localized shadows indicative of infection. The man is clinically well. He should be told that he has some dust in his lungs and to report for another examination in a year. This should be done to be certain that no infection is developing. Such advice will allow the man to continue to earn his living at his usual trade. He already has a disease that is slowly progressive and little more damage will result from further exposure. To advise otherwise makes him neurasthenic and often places him in the bread line, for others may not care to employ him.

The person with localized conglomerate lesions frequently has symptoms and cannot work as hard. A lighter job should be found for him, preferably with the same industry where he has worked and perhaps developed his disease. To discharge him would invite a suit for damage. If he suffers in loss of income, it is fair that he be compensated.

Where symptoms and incapacitation are due to active infection the individual cannot continue in employment. If the case is one of open tuberculosis with bacilli in his sputum he is a source of danger to his fellow workmen. He should be hospitalized and given compensation. If his infection is inactive and he has not yet become incapacitated, light work may be found for him in parts of the plant other than those where dust is generated. If he is a young man with an active focus of infection, hospitalization may afford some benefit. Although it is not generally recommended, the writer feels that he personally would wish to give treatment a trial.

Now that the hazard is being recognized, it is the obligation of industry to prevent the formation of dust. But in some operations the production of a certain amount of dust is apparently unavoidable. Where this is the case the writer believes that it is unsafe to emp'oy a young man (under the age of 30) with any roentgenographic evidence of tuberculosis no matter how well healed his lesion may appear. As to older men there is some conflict of opinion; some authorities think that the healed focus will attract an undue amount of dust and lead to the formation of a conglomerate focus of slicosis that is a potential source of disability. But in a clean plant where the dust concentration is below the danger level, the healed focus of tuberculosis, in the writer's opinion, confers immunity against eexogenous reinfections. The old focus, after this age, is quite apt to be sterile and incapable of reactivation. More experience in following these cases is obviously needed before definite rules of guidance can be formulated.

Relationship of Asbestosis and Silicosis to Disability

THE discussion of this interesting but rather complicated subject may best be preceded by offering acceptable definitions of the factors under consideration.*

The committee on pneumoconiosis of the industrial hygiene section of the American Health Association gives the following definition:

Silicosis is a disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present), and by characteristic x-ray findings.

A similar definition for asbestosis would define it as a disease due to the breathing of air containing asbestos particles, characterized anatomically by symmetrical and diffuse fibrotic changes throughout both lungs, and clinically by shortness of breath, decreased chest expansion, cardiac embarrassment, lessened capacity for work, absence of fever, increased susceptibility to pulmonary infection (some or all of which symptoms may be present), and by positive roentgenological evidence of generalized pulmonary changes.

The term "disability" as used in this discussion may be defined as a decreased capacity to do the work required of the individual in the course of his usual occupation and/or an increased susceptibility to respiratory infection causing a loss of time from work which may reasonably be considered as primarily the result of the pulmonary fibrosis. Disability attributed to simple fibrosis resulting in a decreased capacity for work usually is considered permanent and continuous. In cases of early silicosis in which there is no decreased capacity for work, there is an increased susceptibility to respiratory infection that intermittently may result in disability. When tubercle bacilli are the cause of the infection, the disability is usually considered total and permanent.

Attention is called to the fact that the definitions stated do not specify that a diagnosis of silicosis or asbestosis necessarily means disability. Both are slowly developing conditions and clinical and roentgenological evidence of pulmonary fibrosis may in some cases exist for several years prior to the demonstration of any decreased capacity for work.

The information available from the literature on methods for determining the disability due to silicosis or asbestosis is limited. Particularly is this so in the case of asbestosis. Therefore, the discussion of this subject is necessarily based chiefly on data relative to silicosis.

In the absence of infection, disability due to the pumonary fibrosis associated with silicosis is primarily dependent upon loss of functional lung tissues. This replacement of alveolar tissue by scar tissue leads to decreased oxygenation of the blood. Not only does the abnormal fibrosis decrease the available alveolar space for the interchange of gases, but such spaces as remain function less efficiently, due to the loss of normal elasticity of the lung and delayed inflation and deflation.

The embarrassed heart action is likewise dual in character. There is an increased load on the right heart in forcing blood through the fibrosed lung; and the pulse rate, to maintain efficient circulation during exertion, is necessarily abnormally increased in Nature's effort to get the most from the limited alveolar space available.

It has been suggested that the excessive absorption of silica or asbestos over a long period of time may exert some toxic influence upon the various body tissues, but to date sufficient data are not available regarding such action to measure the specific effect produced. Such conditions as nephritis, arteriosclerosis, digestive or nervous disturbances have not been shown to be produced or to be measurably affected by the action of these compounds.

Disability associated with cases of asbestosis and silicosis which are complicated by infection is not only usually more pronounced but also offers a more difficult question for analysis.

Studies of workers in occupations entailing an exposure to silica dust have shown an increase in loss of time, due to respiratory infection, when compared with similar groups in nondusty atmospheres. Including tuberculosis as well as the common respiratory infections, the data furthermore suggest that the severity of the infection is increased in about the same degree as the incidence. In the granite industry it was found that the severity, when measured by the duration, of cases lasting 8 days and longer, for the group of hand pneumatic cutters exposed to high concentrations of silica dust, was about four times as great as the same for a group exposed to the minimum amounts of industrial dust.

While it may not be practical in the individual case to determine the excessive incidence and severity of respiratory infection which may be attributed to the presence of fibrosis resulting from silica or asbestos exposure, a reasonable estimate may be established through consideration of data collected on specific occupational groups. The following table shows the reported incidence of attacks of disabling respiratory disease in two groups of anthracite coal miners. (Table 1.)

The increased susceptibility to tuberculosis particularly manifested by those with silicosis constitutes a marked potential disability. . Available literature cites but few cases of death resulting from silicosis or asbestosis uncomplicated by infection. Gardner has stated that 75% of those who contract silicosis die of silicosis complicated by pulmonary tuberculosis. In the observations made upon hard-coal miners, it was found that nearly 40% of those with anthraco-

* Dr. R. R. Sayers, at the 1935 Convention of the International Association of Industrial Accident Boards and Commissions, published in "Discussion of Industrial Accidents and Diseases" Bull. No. 4, U. S. Department of Labor, Division of Labor Standards.

silicosis who had worked in rock a goodly portion of their time for a period of 25 years or more manifested evidence of pulmonary tuberculosis as a complication. The following table by Britten, based upon the report of the Registrar-General of England for the years 1921-23, indicates the increase in mortality from pulmonary tuberculosis among various trade groups, those exposed to silica dust showing the highest rate. (Table 2.)

Group	Total Number	Number	Percent
All without anthraco-silicosis	2,095	1,049	50.1
All with uncomplicated anthraco-silicosis	510	351	68.8
All with complicated anthraco-silicosis	106	75	74.5

Table 1.—Number and percent of anthracite miners at work, giving a history of repeated attacks of disabling respiratory infection.

To consider satisfactorily the relationship of silicosis and asbestosis to disability in the individual case, the following information is helpful if not prerequisite:

1. A record of previous and present occupational exposure.
2. A record of past illnesses and present complaints and illnesses.
3. Physical x-ray, and laboratory data to establish a diagnosis and demonstrate disability.

In cases complicated by infection, repeated examinations to determine the nature and severity of the infection may be necessary. Repeated examinations may also be necessary to determine progress of the condition in individual cases.

In recording data relative to occupational exposure, it is important that the record be complete. It should reveal the industries in which the individual has been engaged throughout his working life, and the time spent in each specific occupation within the industry. If the records begin with the present job and list the time spent at such work in the present industry, and follow that with the same data for the preceding job, and so forth, back to the time the individual first started to work, the information will probably be relatively accurate. The following figure illustrates a form which proved useful in collecting data upon a group of hard-coal miners. Slight changes in this form might be made to make it serviceable for other groups (Fig 1).

The past medical record of individuals examined for silicosis or asbestosis should be as complete as possible, especially as regards present respiratory infection. Since the question of disability invariably enters the picture, illness or accidents likely to lead to disability should be revealed.

Present complaints, which are volunteered, are usually of more importance than those obtained by direct questioning. Here again those referable to respiratory infection are especially helpful.

The physical examination should be adequate to discover gross evidence of any body changes likely to contribute to disability. Detailed examination of the chest is necessary. Although early in the development of generalized pulmonary fibrosis clinical manifestations of the condition are usually slight, nevertheless close inspection, palpation, percussion, and auscultation will usually elicit evidence of pulmonary changes in cases showing any decreased capacity for work. X-ray examination of the chest is indispensable. While a good film may serve the purpose, at the same time fluoroscopic examination is helpful in the absence of stereoscopic films. It also reveals information relative to diaphragmatic excursion and the presence of pleural adhesions.

Occupation:	Mortality rate (standardized)
All occupied and retired males	149.6
Tin and copper mines, underground workers not superintending staff	1,886.0
Tin and copper mines, not superintending staff	1,323.5
Grinders in the cutlery tools	1,178.5
Metal grinders	636.7
Slate masons and slate workers	512.5
Potter's millworkers; slip makers, potters	411.4

Table 2.—Standardized mortality from respiratory tuberculosis in occupations with rates above average, males age 20-65, 1921-23, England and Wales.

Repeated microscopic examinations of the sputum, and when negative for tuberculosis, animal inoculation of the material, will sometimes establish a diagnosis of complicating infection earlier than may be accomplished otherwise.

An exercise test in the majority of cases will serve to detect evidence of a decreased capacity for work and its cause. A test, which has proven helpful in examining groups of workers for evidence of silicosis and associated disability, is carried out as follows: The individual places one foot upon a chair or firm stand, 18 inches in height, and raises his body to an erect position 25 times in 30 seconds. Those with marked respiratory or cardiac disturbance cannot be subjected to such a test, but in the course of examining persons already employed or applying for work few of such individuals will present themselves. The pulse and respiration are taken with the examinee at rest, immediately following the exercise test, and after a 2-minute rest period. It is evident that such a test is not one whose results are affected by respiratory conditions only, but one which is influenced by various factors, such as weight, heart condition, age, general physical condition, and numerous metabolic differences. It does serve as an indication of decreased capacity for work. When we consider all factors, definite information concerning respiratory capacity is available. Regardless of the presence of slight cardiac defects or changes due to age, and so forth, the individual with appreciable pulmonary fibrosis will not only exhibit shortness of breath following the exercise, but the altered respiratory rhythm is characteristic. The expiratory phase is markedly prolonged, often to such an extent that the rate of respiration is much less than in the case of a person with pulmonary infection or cardiac disturbances. Due to loss of elasticity of the lung, the individual cannot empty his lungs rapidly enough to allow for a great increase in respiratory rate. In fact, the rhythm approaches that or the asthmatic before exercise. The condition may be differentiated from bronchial asthma by the absence of the râles and other clinical data. In cases of well-established silicosis, prolonged expiration may be elicited by careful observation during the course of physical examination, prior to any exercise test.

The role played by cardiac deficiency may be determined by considering the records of the pulse before, after, and following the rest period, and the blood-pressure determination.

Although the factor of increased susceptibility to respiratory infection may seem difficult to interpret, as a disability, by far the majority of

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cases where the same is demonstrated will have to do with tuberculosis as a complication.

The individual suffering from silicosis complicated by active tuberculosis is truly disabled and should be removed from futher exposure both for his own good and for the protection of his co-workers.

When a diagnosis of pulmonary fibrosis resulting from occupational exposure has been established, the next most important facts to learn are: Is the condition complicated by infection, and is there evidence that the individual has lost any capacity for work?

Often, for purposes of description, particularly when considering the subject of disability in its relation to silicosis, it may seem more convenient to classify the degree of development of pulmonary fibrosis in stages. In this country the most common practice has been to speak of this degree of development as first, second, and third stage.
regardless of other clinical evidence.

From the viewpoint of disability, those in the first stage are usually grouped so because their working capacity is not noticeably impaired.

Those in the second stage may evidence some decrease in capacity to work as well as formerly, while the working capacity of those classified as in the final or third stage is considered seriously and permanently impaired.

Diagnosis of Silicosis

SILICOSIS is, without doubt, the easiest of all occupational diseases to diagnose.* In the first place, it seems never to occur except through industrial exposure, as there is no evidence to the writer's knowledge that domestic life has ever provided enough exposure to incur the disease. Again, when the disease-picture is once established, it "stays put"—its tell-tale markings do not fade out or disappear, so that advantage can be taken of the "wait and see" policy whenever doubt exists. Thirdly, in chronic silicosis, nodules are invariably found by the time the affliction has become disabling.

Dismissing corrosive and directly poisonous dusts, the lung effects of breathing any dust are termed pneumoconiosis (lung-dust-disease). At present, the effects of any dust are considered as active (proliferative), inactive (inert), or absorptive (the dust is absorbed or disappears).[1] An active dust produces scar-tissue (fibrosis) in its immediate vicinity, which effect occurs when the dust particles are stopped in transit. This stoppage occurs first at the end of the lung drainage system, i.e., chiefly in the lymph nodes surrounding the lower part of the wind-pipe (trachea) and its larger subdivisions. A "backing-up" effect next follows and the dust accumulates in the drainage channels (lymph ducts) which come from all parts of the lungs.

Apparently, only siliceous dust is active, and the scar-tissue laid down in the drainage ducts, at first as strands, soon "balls up" producing strings of beads and, finally, isolated knobs known as "nodules." Both beads (or "beading") and nodules show in properly taken x-ray pictures. Beading may occur in other affections but nodules are characteristic of silica effects.

Likewise a local, blotchy condition known as "mottling" often occurs in infectious like tuberculosis, as well as what may be termed "pseudo-nodles" from several causes, but other circumstances and the "wait and see" policy—a matter usually of a few months at most—rather easily differentiates these conditions (see below).

This definition of the disease Silicosis is given by the Committee on Pneumoconiosis, Section on Industrial Hygiene, American Public Health Association:[2]

"Silicosis is a disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present), and by characteristic x-ray findings. The disease is divided arbitrarily into first, second, and third stages for convenience of description and possible compensation purposes."

To this the Committee added, at its Milwaukee meeting in October, 1935, the x-ray-pathological differentiation of stages (i.e., of the chronic or usual form of the disease), as devised by a special committee of experts.[3] Stages merge from one into another without sharp distinctions.

The presence of nodules (nodulation) in both lungs is fundamental to diagnosing silicosis, whether in x-ray pictures, animal test-experiments, or at autopsies on human lungs.

Acute silicosis, the much rarer form, results when enormous amounts of fine, siliceous dust become rapidly imbedded in the lungs. Thus within a few months to a year, distressing symptoms, chiefly respiratory, may appear almost before the x-ray shows anything definite.[4] Very soon, however, a bilateral "haze" appears in x-ray pictures, usually in the lower mid-lung regions (the parts most used for breathing purposes), and, as it spreads, soft nodules come to occupy it. The term subacute silicosis might well be applied when the disease makes its first appearance after a year of exposure and under five years. Acute and often fatal pneumonia is a common complication of intense silicosis.

Date ..... Office .....

Hospital .....

Name ..... Present age .....

Age began work..... Number of years worked.....

	Specific occupation	Specific industry	Number of years in—		
			Hard coal	Other dusty	Non-dusty
Present					
Preceding present 1.....					
2.....					
3.....					
4.....					
5.....					
6.....					
7.....					
8.....					
9.....					
10.....					

Remarks:

Recorded by.....

Fig 1.—Occupational Record

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* EMERY R. HAYHURST, M.D., Consultant on Occupational Diseases, Ohio Department of Health; published in *The American Labor Legislation Review,* preceded by the following EDITOR'S NOTE: "All-inclusive occupational disease compensation has been delayed in many states by assertions of the opposition at public hearings that it is impossible to diagnose silicosis. Those who have most persistently made this claim have not always been disinterested witnesses. But such allegations, frequently repeated, require attention because simple justice to all victims of occupational disease should not be further delayed through misapprehension of the difficulties. An authoritative statement is needed. Outstanding among those who have for many years specialized from the scientific approach in this field is Dr. Hayhurst, who by medical training and exceptional research and experience, occupies a position of unquestioned authority. He has been several times chairman of the Industrial Hygiene Section of the American Public Health Association. His scientific articles and books upon the subject have given him an international reputation. At our request he has written for this REVIEW a concise statement on the specific point of the practicability of silicosis diagnosis."

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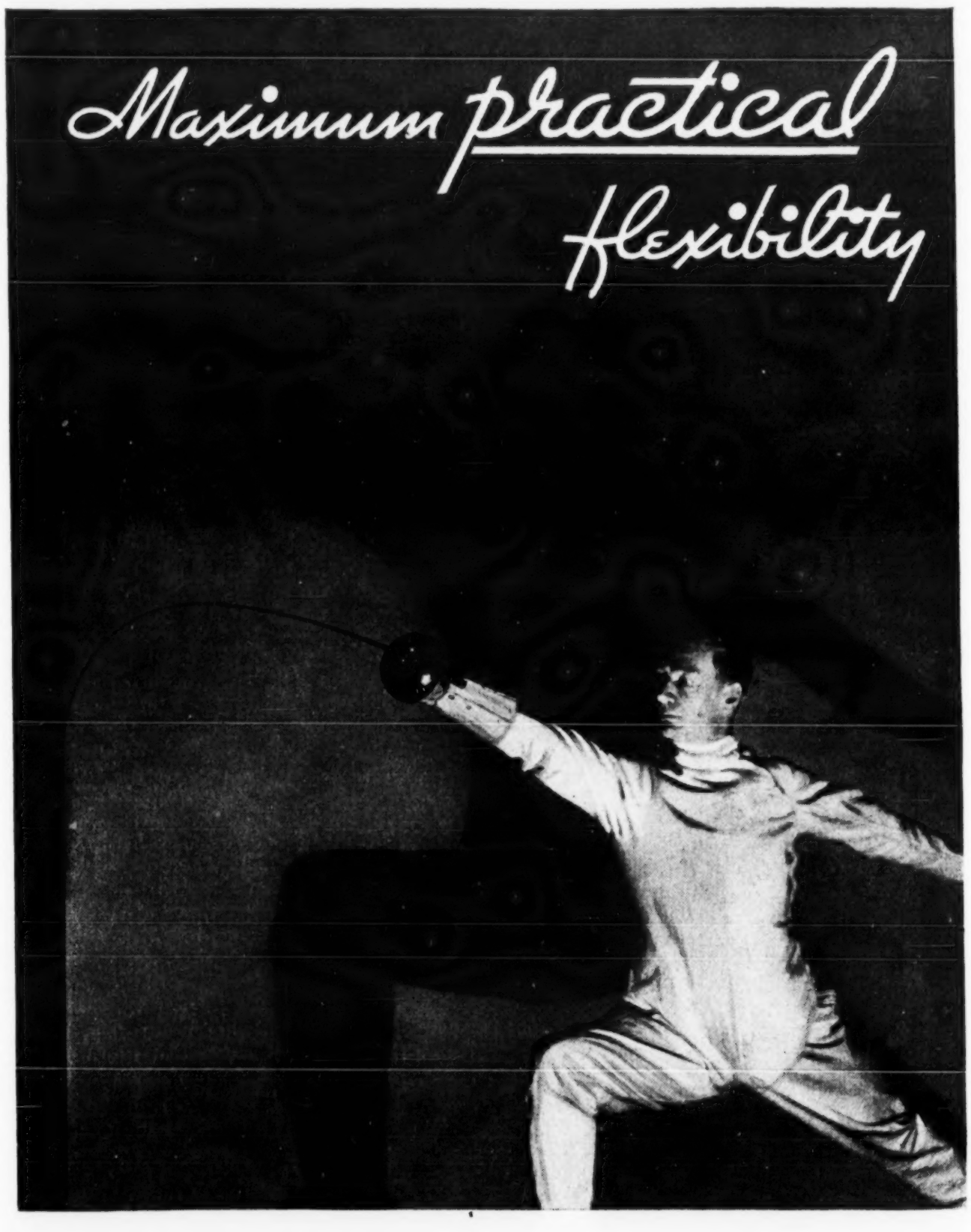

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The several features necessary to diagnose chronic silicosis may be summed up as follows:

(1) Exposure to silica dust: In all cases where the disease, in chronic form, has been found in man, the exposure has been from about five to 20 or more years, and to amounts of silica dust greatly in excess of anything known to occur in nature. The amount and fineness of the dust, the higher the percentage of silica (in a mixed dust), the buoyancy of the dust, and its degree of dryness, are major hazardous factors. Certain standards, such as the number or weight of dust particles per cubic volume of air inhaled, have now been tentatively agreed upon as fixing the limits of quantitative exposure.[5] Prior employment in a mineral dust (including coal dust) seems to predispose the lungs, probably by partial blocking of normal drainage outlets.

(2) Thanks to an early observation of Sayers and his co-workers[6] at the U. S. Bureau of Mines, a definite test for proliferative, nodule-forming dust is now available. Small amounts of the suspected dust are injected into the peritoneal cavities of guinea pigs. In 30 to 90 days, growing nodules are definitely formed, in the case of siliceous dusts.

(3) Nature of work: Hard work, high temperatures, dry atmospheres, the presence of abnormal gases or decreased oxygen (as in mines, tunnels, trenches, or at high altitudes) —all necessitating deeper breathing —likewise, long hours and absence of holidays, are important predisposing factors.

(4) Personal factors: The efficacy of the nose as a dust filter, middle age or beyond, mouth-breathing, close proximity to the source of dust, carelessness in personal precautions, diseases of the lungs or other organic and constitutional diseases (which usually interfere with respiration)— all enter in as important factors.

(5) X-ray findings: In perhaps four out of five claims, x-ray findings alone will establish the presence or absence of silicosis. "No nodules, no possibility of silicosis", at least at that "reading" Later x-ray pictures may show them, since they develop slowly. Hence the value of serial pictures, taken about every six months to a year. The nodulation of silicosis is quite characteristic in itself — location avoiding the periphery, quite symmetrical distribution, the shape, size, density, discreteness, permanency, trend toward conglomeration, etc., of the nodules. Space does not permit describing these. At least one pair of stero-pictures should be studied where any doubt exists. Different forms of silicosis dusts may vary the x-ray picture, so that silico-anthracosis, silicosiderosis, silicatosis (the latter including asbestosis), and various mixed forms, occur.

Since it is obvious that there must be some pathology in the lungs before nodules appear in silicosis, which pathology exhibits increased fibrosis, older nomenclatures described this phase as the ante-primary stage. As such, it could never be positively diagnosed since many other conditions may produce the same picture. Hence, these types of cases should never be brought into court as definite silicosis. Chronic silicosis is also without disability in this phase.

(6) Physical examination of the patient shows nothing characteristic, but suggestive are: decreased chest elasticity, pulmonary ventilation, and breath-holding capacity; likewise impaired resonance and breath sounds. Clubbed fingers, curved finger-nails, and cyanosis may develop later, showing circulatory-respiratory embarrassment. The pulse is rarely above 100, while temperature remains normal, unless infection is a complication.

(7) The patient's complaints: These are usually absent in the first stage and only nominal in the second. As the years go by, dyspnosa, cough, expectoration (usually not pronounced unless the case is complicated), pains in the chest, malaise, sweating, anxiety, disturbed sleep (coughing), vomiting on effort—all may occur. Fever means infection.

Space does not permit a discussion of differential diagnosis, but, in general, simulating conditions include symmetrical bi-lateral blotchy fibroses, tuberculosis (especially the miliary or "galloping consumption" type), and mottling from various (usually infectious) causes. Multiple calcified foci from childhood-type tuberculosis, a very common condition in adult lungs, are usually limited to a relatively few, irregularly distributed, densely opaque nodules which are easily differentiated, if not by the x-ray, then by considering all the factors involved. Calsified mycotic lesions, such as healed abscesses from various spore infections, have only to be mentioned; likewise, artifacs resulting from faulty technique, inadequate illumination of the x-ray picture, misleading normal markings (criss-crosses and axial views of blood vessels), etc. Silicophobia, malingering, exaggeration, etc., are quite easily ruled out, either by the x-ray or other features.

Finally, absolute diagnosis, it is commonly accepted. may be made upon post-mortem findings, when the chemist will show more than 1% of silica in the ash of the completely dried lung,[7] and the mineralogist can demonstrate characteristic birefringent particles under the polarmicroscope.

In conclusion, a certain diagnosis of silicosis cannot be made upon any one of the single factors discussed, but necessitates a consideration of several, especially the nature of the exposure, the x-rav findings, and the progress of the disease. As a last resort, a doubtful case should be held over for a few months to watch developments when simulating conditions and circumstances invariably eliminate themselves.

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Low Back Pain

THE literature of orthopaedic surgery and the private conversation of orthopaedic surgeons is becoming increasingly voluble with the subject of low back pain.* Committees are formed from the American Academy of Orthopedics and from the American Orthopedic Association. I presume they meet and discuss, at least they issue reports, reports which urge the value of further work on this vexed problem. Two concepts in general hold sway, the mechanical, the infectious or toxic. Of recent years Albee has been preaching that most so-called lumbago is a myofibrositis. I had occasion to lunch beside him a few years ago and heard it all expounded with vigorous conviction and mallet stroke diction, but I was left a bit vague as to whether this myofibrositis is due to focal infection, the phases of the moon, or the Hoover administration. This after all is but a revivification of an old concept and rests on a solid foundation of cases miraculously cured by tonsillectomy, by prostatic massage, or by tooth extraction. "The cause of backache" say these people "is anywhere but in the back." The mechanical concept of backache is, I think, attributable to Goldthwait who about 1903 published an article about the sacroiliac joint. He has had occasion to repent very deeply that he did so, for since then sacroiliac subluxation or strain has been the diagnosis put on thousands of cases which show tenderness to pressure over the posterior superior spine, or show a limitation of straight leg raising on one side. And yet I believe he was right in his fundamental unspoken conviction that the cause of backaches is in the back and that it is there that the cause must first be sought, though of later years he and his followers have paid less attention to the sacroiliac and more to the lumbo-sacral articulation. The exercises of Mennell, the sacroiliac fusion of Smith-Petersen, the facetectomy of Ghormley, the lumbo-sacral fusion and trisacral fusions of many others are implied acceptance of the mechanistic viewpoint. This paper is an attempt at an exposition of that viewpoint.

You see we are well built to run on all fours; we gained immeasurably when some remote arboreal ancestor first stood erect, but we have paid for our gain in backache. For an animal whose femur is at right angles to his spinal column to stand erect is to extend the hip to the very limit of tolerance. Now joints do not easily stand being stretched to this

*Robert A. Milliken, M.D., Little Rock Arkansas; read before the Sixty-first Annual Session of the Arkansas Medical Society, Hot Springs National Park, April 29, 1936; published in *The Journal of the Arkansas Medical Society*, July, 1936.

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limit of tolerance and so to ease the hip situation the pelvis went only half way, forcing the lumbar spine to take up the rest of the motion. That left the weight of the trunk supported on a sacrum slanting at a 45° angle and on the posterior architectural of the lumbar spine, articular facets, and spinous processes, crowded together unduly. To add to the damage the whole pull of gravity on the viscera was changed and to maintain the quadruped relationship requires a high chest and a tight abdomen quite impossible to most of us. When these things go the pull of gravity on the viscera is transmitted through a chain of suspensory ligaments (described by von Teuteben in the seventies, but since neglected) to the base of the neck. That means that visceroptosis drags the head forward which in turn demands an increase of lumbar lordosis to compensate and keep the head over the feet. Consider now these structures affected by that lordosis. The spinous processes particularly of the fourth and fifth lumbar vertebrae, and sacrum are crowded together until they actually impinge one against the other, and I have proved at operation that there may develop a psuedarthrosis in which arthritis can arise. Then the ilio-lumbar ligament, one part of which runs from the fourth lumbar to the crest of ilium, is put upon an added stretch in response to which it thickens. Thirdly, the lumbo-sacral articular facets are so moved that this joint is put to an extreme stretch. Now a joint is that is put to an extreme stretch for a long period of time is certain to become sore, to cause pain, to have periarticular thickening and to develop hydrops. If it be remembered that the edge of this joint forms part of the wall of the intervertebral foramen and that the largest of the lumbar roots, the fifth, passes through this, the smallest of the lumbar foramina, we can understand why swelling of the joint could cause nerve pressure with pain referred along the sciatic nerve formed, as you recall, from the fourth and fifth lumbar and the first, second and third sacral roots. If, as so frequently happens, there is a degeneration and narrowing of the intervertebral disc then the crowding together is accentuated. At the risk of being repetitious let me illustrate in another area. If any one of you were to put a two-inch wedge under the median edge of your shoe so that you walked all day on the edge of your foot, you would not be surprised if your ankle became sore. As the strain continued you know that it would get so sore as even to ache at night and you forsee all sorts of adaptive changes that would occur in such a foot. Now if you were to go to bed for a few days and rest that foot you know that most, if not all, of the pain would subside to return again as you resumed walking on the wedge. Now increased lumbar lordosis in the spine is exactly comparable to a wedge under the foot. Cure in either condition is to be had only by removing the mechanical abnormality. But, you will say, most adults have poor posture and not all have backache. True, and not all club feet have continuous pain. But you know that such feet are prone to strain and if called upon to treat a painful club foot you would endeavor to remodel it to normal relationship. So with backs, the changes take place so slowly that adaptive changes can occur. Such backs, however, are at their limit of tolerance and have lost their margin of safety so that any slight over-use, or fatigue, tips them over.

Now, of course, there are other causes of low backache, the commonest being congenital anomaly, tuberculosis, malignancy and arthritis. All of these can be found by physical examination supplemented by x-ray study, which should always include a lateral film. Indeed, no spine can be said to have had an x-ray examination unless it has had a lateral study. The differential diagnosis between compression fracture, tuberculosis and malignancy is impossible except in rare cases on the anteroposterior film, while such anomalies as spondylolisthesis, and such conditions as Buchmann's epiphysitis, Kummell's disease, or Schmorl's disease would be altogether missed.

The treatment of the postural cases is sometimes startlingly easy, sometimes it tries the patience of every one to the limit. Obviously the treatment of a mechanical difficulty is mechanical, a correction of the mechanical error. First come exercises to strengthen the relaxed abdominal, dorsal and gluteal muscles. Where the factor of safety has been only slightly overstepped a surprisingly slight increase of muscular tone and change in the pelvic tilt will work wonders. Many cases need more than that, they must have rest for the underlying fatigue and rest for the irritated ligaments or periosteal areas; then, because few people can afford the necessary weeks in bed and must be up and doing artificial support is needed, a back brace or a corset. The immediate relief that many a case gets by the addition of external support is striking. Many cases need stretching of dorsal kyphosis, or drooping ribs, of flexed hips, before the muscles can get the body into the proper posture. So then these cases need exercises, rest, stretching and braces in various proportions and amount, but always and forever exercises. One does harm rather than good by stretching without strengthening; bracing alone weakens, rest alone weakens. Lastly, do not forget the feet. Footstrain may not have been the straw that broke the camel's back, but it removal will often relieve. The late Dr. Lovett used to say that posture begins at the floor. The bad foot mechanics and the bad back mechanics are interrelated and interdependent. We cannot cure the one if we ignore the other. One other point, measure the legs. A surprising number of backaches have been cured by adding a ⅜ inch lift to one heel.

Treatment of the diseased or anomalous cases depends on an appreciation of what it is that is causing the pain. Here again, whether it be spondylolisthesis or arthritis, it will usually be found that it is abnormality of motion that is the pain factor. First, we would try to eradicate the abnormality, and if that failed as it usually does, we would abolish the motion by fusing together those vertebrae between which motion is at fault. Inasmuch as the treatment of tuberculosis is rest, rest, and yet more rest, it is in this condition that spinal fusion has its most positive indication.

You will note that nothing has been said about searching for focal infections and their eradication, nor about retroversion of the uterus. It is not to be denied that these and other remote causes can result in backache. You have all seen them and so have I. Indeed one of my colleagues is still joshing me about a backache that persisted under my ministration and which yielded in his hands to dilation of an anal stricture. But, in my opinion, it is pernicious practice to assume at the start that such a factor is present and in the search neglect to consider the back itself. I recall a case of spinal caries with kyphos subjected to uterine suspension and a case of anomaly with terrific sciatica treated by internal medication, and many others. It is certain that if uterine suspension is needed a pessary will give relief and be a therapeutic test that may save a useless operation. It is certain that to try mechanical means first and then resort to operative measures if they fail is easier on the patient than to subject him to a useless operation and then come to the mechanical measures.

I have said three things I should like to have remembered:

1. Look for the cause of backache in the back.
2. Take a lateral x-ray view.
3. Assume the case to be postural if no other pathology is found.

The Relationship Between the Industrial Surgeon, the Insurance Company and the Employee

THE passange of the compensation laws in the different states brought into existence insurance companies which are writing compensation insurance, and presented us with the problem of the injured employee who knew he was going to get compensation and tried to secure more than he was entitled to. *It has also created the industrial surgeon who acts as a buffer, if you wish to call it such, between the insurance company and the injured employee.

I think possibly I will say a few words first in regard to the insurance companies. Naturally, they are interested in having physicians who take care of their work to be those who have had a certain amount of training in handling industrial cases. Therefore, they expect certain definite treatment for their injured employees. They expect them, the physicians, to answer all calls as promptly as they can, make a very thorough examination, determine whether that patient shall be sent to the hospital or whether he can be treated at home, or whether the patient can come to the office for treatment. They also expect the attending physician to take x-ray pictures whenever it is necessary, and to make such check-up x-rays as are

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* William C. Goenne, M.D., F.A.C.S., Davenport; presented before the Eighty-fourth Annual Session, Iowa State Medical Society, Davenport, May 8, 9, 10, 1935; published in *J. Iowa M. Soc.*, July, 1936.

required. However, they assume that the attending physician will not take more pictures than are necessary. They also expect a certain amount of laboratory work and an adequate number of dressings for any injured employee. If the patient is sent to the hospital, they naturally feel that the attending physician will discharge that patient as soon as his condition warrants. In other words, summing up the attitude of the insurance companies, they expect the attending physician to return this man to his employment in the best physical condition possible, in the shortest length of time, and with the least possible expense.

The injured employee, on the other hand, has an entirely different viewpoint. He thinks first of all, "How long am I going to be off?" He doesn't, at the time of injury, take into consideration the severity of his injury or the difficulty which the attending physician is going to have in repairing that injury. After the physician has done all that he possibly can for this injured employee, he begins to think, "How much compensation am I going to get?" and when the doctor tells him it is possibly ten per cent or fifty per cent, in very many cases he would like to get at least twice that much, if possible.

At this point the physician enters, acting as a buffer between the insurance company and the injured employee. It is of paramount importance for the physician to be impartial and forget the fact that the insurance company is going to pay the charges. He must make a report commensurate with the amount of injury or amount of disability that resulted. I recently had occasion to examine a man, and I reported a 75% loss of function in his leg. The insurance company offered this gentleman 30%. That illustrates very clearly a physician's position. The insurance company is dissatisfied with you for estimating a 75% disability when they think it should be 30%, and the man is dissatisfied with 75%, when he feels that he should get 100%.

In conclusion, I hope you will bear in mind that a very definite relationship does exist between the industrial surgeon, the insurance company, and the employee.

Discussion

DR. FRANK M. KEEFE, Clinton: The scope of this subject is of such magnitude, that in the few moments allotted to me I can only dwell on the salient points relative to the relationship between the industrial surgeon, the insurance company or carrier, and the employee. The importance of it should force the attention of every physician who elects to treat industrial cases.

The physician who has a liking for industrial practice and whose contact shows a gradual enlargement soon finds himself devoting his full time to his endeavor, although not full time in any one corporation. The physician or surgeon dealing with diversified industries rapidly develops a wealth of experience.

Fundamentally, all employees are entitled to the same standard of medical supervision and service, irrespective of the size of the company. The majority of employees do not know the compensation law. Notices, properly displayed, of the compensation act would be of great value to the employees as well as the employer. Employees should be sent to a designated physician, and a workingman should not choose his own physician. This practice, I believe, is dangerous. Although the man may be qualified as a family doctor, he may not be qualified as an industrial surgeon, and the reverse, of course, is true. The compensation should be thoroughly explained to them. They are frequently misled by false information which results in legal procedure, generally resulting in the loss of position and money. Trivial as well as major injuries should be reported. Too much cannot be expected of the average employee whose chief aim is making enough money to support his family.

As for the insurance company or carrier, by far the ideal arrangement is the self-insured, and, from the surgeon's standpoint, this plan is carried out by a great many of the railroad trunk lines under the direct supervision of a chief surgeon who, in turn, reports to the claim department. They comply with the various state compensation acts, and their settlements are, as a rule, satisfactory with the disabled employee. Their safety first plan has worked to an advantage to both the employee and the employer. As for the mutual and stock companies, we have all had our pleasant and unpleasant experiences with them; all the way from cutting your fees by some subordinate trying

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to make a name for himself, to questioning you as to your professional ability, honesty or judgment on the severity of the case, whereas if they would conduct their own department and let the surgeon work with them conjointly, the whole situation would be more harmonious and probably more economical.

The insurance company has its troubles, too, because we have been careless in not reporting cases early and not being specific in our reports, description of injuries and the estimates of disabilities, and giving subsequent reports on the progress of the complications. Our profession should sponsor an educational program to appraise a corporation from the standpoint of dividends which adequate medical service pay, and if industry makes the demands, surely the medical profession can meet them, as we have in the past.

Assuming the inherent right of the carrier and the employee, what, then, are the essentials of the medical service? Briefly, the duties that an industrial surgeon should be called upon to perform are:

1. The care of injuries, diseases and abnormal conditions covered by the compensation act.
2. The pre-employment examination, and periodical re-examination during employment.
3. The care of the industrially disabled.
4. The eradication of accident and health hazards.
5. The prevention of the spread of communicable diseases.
6. The regulation of sanitation in the factory.
7. Cooperation with the employer in the selection of jobs to fit the physical and mental status of the employee.

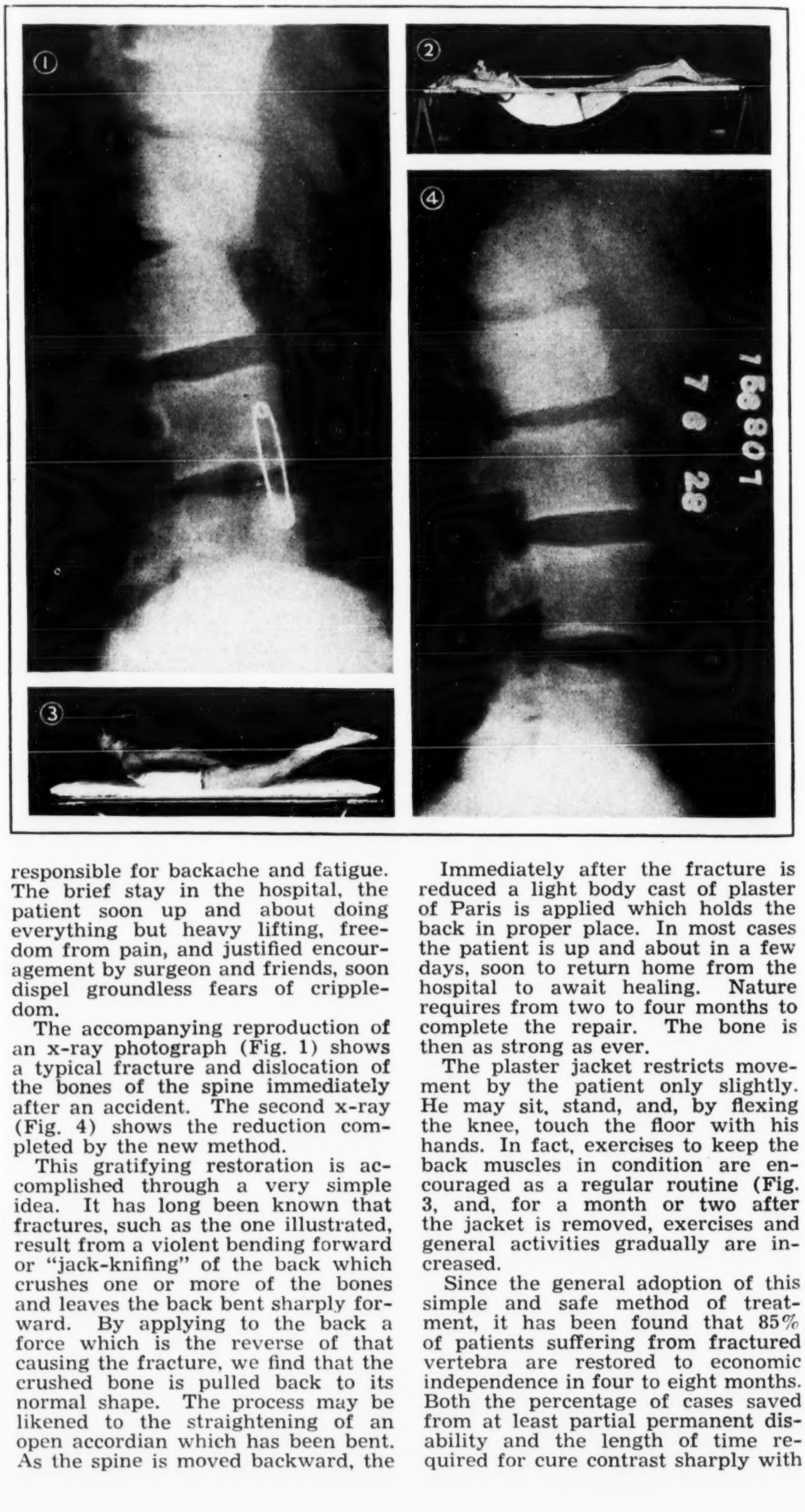
Of the many industrial surgeons, how many do we find performing these duties? Unfortunately, we find that the practice is limited to the care of accidents and disease. Much has been said about the ineffectiveness of th physician in dealing with industrial problems, and there is no denying that there is some cause for this criticism, but far too little has been said about the backwardness of the corporation in recognizing the necessity for complete medical service.

A Treatment of Spinal Fractures

RECENT advances in surgery include the conquest of the problem of the broken back insofar as it relates to bone injury. This is proving to have far-reaching significance to the individual and to the industrial accident budget. Heretofore, the fractured spine of industry has been both an expensive and a demoralizing accident.

Before the development of modern methods of treating spinal fractures long periods of disability for the injured were the rule. Two major reasons were responsible for the long time required for treatment and full convalescence. First, the patient was discouraged or unnerved, either by the severity of his accident, the course of treatment or by the fear of permanent disability. Second, the crushed vertebra could not be reduced to its former shape with the result that the injured suffered backache or tired easily for long intervals following the major course of treatment.

Both of these difficulties can now be overcome. Prompt and complete reduction of the fracture, now possible through recent discoveries, followed by new methods of supporting the back while nature is healing the bone eliminate the factors formerly responsible for backache and fatigue. The brief stay in the hospital, the patient soon up and about doing everything but heavy lifting, freedom from pain, and justified encouragement by surgeon and friends, soon dispel groundless fears of crippledom.



The accompanying reproduction of an x-ray photograph (Fig. 1) shows a typical fracture and dislocation of the bones of the spine immediately after an accident. The second x-ray (Fig. 4) shows the reduction completed by the new method.

This gratifying restoration is accomplished through a very simple idea. It has long been known that fractures, such as the one illustrated, result from a violent bending forward or "jack-knifing" of the back which crushes one or more of the bones and leaves the back bent sharply forward. By applying to the back a force which is the reverse of that causing the fracture, we find that the crushed bone is pulled back to its normal shape. The process may be likened to the straightening of an open accordian which has been bent. As the spine is moved backward, the ligaments pull the fragments of bone into place.

The mechanism for this process is a simple frame. The patient is placed on this just as on any ordinary cot after his body is encased in a felt padding and a plaster jacket. By adjusting the sling on the frame (Fig. 2) the spine is bent backward. So simple is the maneuver that in almost all cases it has been found to be unnecessary to use a general anaesthetic.

Immediately after the fracture is reduced a light body cast of plaster of Paris is applied which holds the back in proper place. In most cases the patient is up and about in a few days, soon to return home from the hospital to await healing. Nature requires from two to four months to complete the repair. The bone is then as strong as ever.

The plaster jacket restricts movement by the patient only slightly. He may sit, stand, and, by flexing the knee, touch the floor with his hands. In fact, exercises to keep the back muscles in condition are encouraged as a regular routine (Fig. 3, and, for a month or two after the jacket is removed, exercises and general activities gradually are increased.

Since the general adoption of this simple and safe method of treatment, it has been found that 85% of patients suffering from fractured vertebra are restored to economic independence in four to eight months. Both the percentage of cases saved from at least partial permanent disability and the length of time required for cure contrast sharply with

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* William A. Rogers, M.D., Consultant, American Mutual Liability Insurance Company, written especially for *American Mutual Magazine* and published in its issue of September, 1936.

former results. The saving in both economic and human values is immeasurable.

There remains, however, a group of back injuries, fortunately small, for which little can be done to repair damaged structures. These are the cases involving injuries to the spinal cord with resulting paralysis. Fortunately nature provides strong protection against injury to the spinal cord, the great nerve substance which passes from the brain down the center of the vertabral column to give off nerves to all parts of the body. Because of this protection the spinal cord is not injured in by far the most cases of back injury. Nevertheless, the fracturing force occasionally not only breaks the back bone but crushes the vital nerve. Such damage is irreparable, for the spinal cord is unable to grow new nerve substance to replace that destroyed in the accident. Under such conditions paralysis and permanent total disability of the injured is the probable result.

While the industrial executive has the prevention of back as well as other injuries as his direct responsibility rather than their cure, he may find some stimulus toward greater effort in his own task of accident prevention in the consciousness that the medical profession is keeping pace with him and is helping reap benefit from his work through savings in human ability and in the medical costs of accidents.

The Industrial Aspect of Hernias (With a Classification and Descriptions)

CONTROVERSY is the essence of progress. In the hernia field, discussions relative to the medicolegal aspect of hernias and about the efficacy of the injection method are creating a large amount of interest.* This is not surprising, since approximately one out of every seven individuals has some type of hernia, and this field is one of the most important, from an insurance angle.

In order to have a clearer concept of hernias, it is advisable for the surgeons to study their pathologic findings at operation and render an indispensable aid to the medical profession by tabulating their findings as to congenital predisposition and traumatic aggravation. The industrial commissions have held that the operating surgeon is best able to determine responsibility in the acquiring of hernias, and as a result most of their findings have been against the industrial interests. The outcome naturally has been a gradual alteration, in removing the responsibility from the employer's shoulders and placing it on the individual's, based on congenital predisposition and on the fact that the worker has sixteen hours at home, against eight hours at work, for acquiring a traumatic aggravation. This move has met with considerable disfavor among the medical profession, due to erroneous concepts relative to congenital predisposition and traumatic aggravation.

The ensuing outline is offered in the hope that other surgeons will advance surgical statistics that will either approve or disprove the present industrial attitude. The ornithologists maintain that everybody has seen an English sparrow, but that few can describe its build and markings. Likewise, many thousands of surgeons have seen hernias, but few have analyzed their operative findings or kept statistical data. A preponderance of hernias in the male are of the indirect inguinal type, so that the major portion of the ensuing outline will deal with this type.

INDIRECT INGUINAL HERNIA: An indirect inguinal hernia is the passing of part of the intestines, mesentery, omentum, or a combination of these to the inguinal region, by way of the inguinal canal, passing lateral to the deep epigastric artery.

CLASSIFICATION: I.—Congenital, resulting from an incomplete clearing up of the tunica vaginalis testis after the descent of the testicle, during the seventh and the eighth months of fetal life.

1.—Infantile, in which a definite hernia descends posterior to the congenital sac.

II.—Acquired.

1.—Acutely acquired, due to:

A.—A poking or pointed injury from the outside, applied directly to the inguinal canal structures, especially those of the internal ring.

B. — Increased intra-abdominal pressure, due to external force or injury to the abdomen, causing increased pressure, or to

C.—Fixation of the abdominal musculature, with increased pressure produced by the forcing down of the diaphragm, as in coughing, straining at the stool, lifting, etc.

2.—Gradually acquired due to:

A.—Weakness or relaxation of the

* RUSSELL A. WINTERS, M.D., in *Clinical Medicine & Surgery*, September, 1936.

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internal ring, with a continuous intestinal barrage against it, or to

B.—Congenital predisposition to a hernia by way of the

a.—Parietal peritoneal self over the deep epigastric artery, forming a flucrum for assault against the internal ring.

b.—Congenital weakness of the wall at the intrenal ring, due to an undemonstrable processus vaginalis pocket.

c.—Plica vesicularis—a fold of peritoneum extending over the bladder and passing beneath the internal rings, supporting this persistent localized traumatizing action.

d.—Obliterated hypogastric artery, persisting to form a direct hernia pocket with an associated predisposition to indirect hernia.

ANATOMIC STRUCTURES INVOLVED: I.—The internal ring is, theoretically, located ½ inch above the inguinal ligament, midway between the anterior superior spine of the ilium and the spine of the pubis. It is an oval opening in the transversalis abdominis muscle fascia, formed by a prolongation of this fascia around the cord in a funnel-shaped manner and passing between the fibers of the muscle.

II.—The inguinal canal is about 1½ inches long, passing consecutively through the three intersupporting muscles, the innermost portion passes through the fibers of the transversalis abdominis muscle, whose fibers run transversely across the abdomen. At the internal ring the supporting sheath is the internal oblique muscle, external oblique muscle, fat, fascia, and skin; the middle portion passes between the fibers of the internal oblique muscle; whose fibers pass downward and outward from the rectus abdominis muscle; and the outer portion penetrates the external oblique muscle, the fibers of which pass upwards and outwards from the rectus abdominis muscle, forming an angle of 45 degrees with the transversalis abdominis muscle.

III.—The external ring is a triangular opening in the aponeurosis of the external oblique muscle, situated just below and external to the crest o fthe pubis. Normally it is about ½ inch wide and 1 inch long. The spermatic fascia of the external ring is composed of a group of tendinous fibers which arch across the lower part of the external oblique aponeurosis, between the pillars of the external ring. These are connected together and continue downwards in a tubular prolongation enclosing the cord and the testis in a definite sheath. This sheath, formed by the external oblique muscle, is known as the cremasteric muscle.

IV.—Congenital Abnormalities include the processus caginalis, a sac left following the descent of the testicle and tunica caginalis, which may persist in any degree, from a slight invagination at the internal ring to a definite sacculation which may extend down the inguinal canal to any lower level, occasionally uniting with the spermatic fascia and enclosing the testicle.

The location of a congenital hernia is most frequently on the right side and is usually associated with a hernia of less degree, or a relaxed ring, on the left side.

Relaxed external rings predispose to hernias, because the transverse abdominal muscle is pushed against a larger opening, causing it to be thinned out at this point and producing a muscular bulging, or causing the thinned muscle fibers to separate, permitting the passage of intestines or omentum.

Relaxation of the inguinal canal muscles, occurring when the patient is in a sitting, lifting, or bent-over position, also contributes to the formation of hernias.

PAIN IN HERNIAS: I.—Pain in acutely acquired hernia would be instantaneous and intense, because

1.—The peritoneal sac is pushed through the internal ring and traumatized. (Peritoneum is extremely sensitive, as are all tissues guarding the vital structures, such as the pleura, pericardium, tendon sheaths, etc. The degree of sensitivity is directly proportional to the importance of the structures protected. Nausea or vomiting may result from this irritation or from the intestinal inclusion.)

2.—The fascia around the internal ring opening and along the cord is torn loose, permitting the passage of the sac.

3.—The muscle fibers of the three inter-supporting muscle layers are torn apart in the three different planes.

4.—The fascia at external ring is torn through, in order to permit the passage of the sac.

5.—Inclusion of the sac and its contents in the constricting traumatized structures retards its circulation. Associated discomfort may persist for over two weeks, because muscle requires two or three weeks for normal repair and the cessation of tenderness. Reduction and recurrence hernia, due to manipulations or changes in positions, produce a constant irritation, which retards the normal "resting tissue repair time," prolonging the tenderness to six or eight weeks or more. Pain radiates from the inguinal canal down the groin, if a nerve is involved.

II.—Gradually acquired hernia may have

1.—No pain with it, because the deep epigastric artery lies between the transversalis fascia and the peritoneum, passing obliquely upward and inward along the lower margin of the internal ring, with its "mesoarterial" sheath forming a pocket just below the internal ring, which supports a continuous intestinal onslaught against the ring. The gradual enlarging of the ring and canal structures might go unnoticed or be evidenced by occasional discomfort when

A.—An abnormally large loop of intestines replaced the smaller one that has been persistently present. This seeming anesthesia to trauma results from a continuous pressure and can be correlated with the gradual stretching of the lips in the Ubangi woman in Africa without much physical discomfort.

B.—Incomplete taumatic hernia of old standing would come under this classification; also enlarging over a period of time, without much physical discomfort. When pain is present in such cases, it indicates the passage of an abnormally large sac content; but the pain does not persist and there is very little tissue trauma associated with its presence, since the stretching of the tissues has been progressing over a variable period.

Congenital hernia would produce no pain if the condition has not changed since birth; or occasional discomfort, as in a gradually acquired hernia.

CONDITION OF THE SAC: Normal peritoneum forming the sac suggests a comparatively recent affair, or an older condition in which the small sac has been permitted to pass through a large ring without any associated irritation. Peritoneum is not normally attached to the abdominal wall and should not be altered on being pushed through the ring.

Thickened peritoneum forming the sac suggests an old affair, in which a series of passages through a comparatively tight ring have produced a chronic irritation with thickening.

Thinned peritoneum forming the sac suggests either

I.—Adhesions of the peritoneum at the internal ring or in th ecanal, preventing the addition of more peritoneum as the size of the sac is increased by pressure and a natural enlargement of the sac results from the hernia content pressure, or

II.—Congenital processus vaginalis, evidencing itself by hernia formation, its walls being adherent to the inguinal canal and thinned out as in the above case and its development being retarded by lack of use until hernial contents are introduced.

A sac, present but not palpable, may occur where the sac is adherent to the walls of the canal and the contents of the hernia are retained within the abdominal cavity by a temporary valve-like action of the fibers of the transversalis abdominis muscle, or a fold of peritoneum acting as a valve. This explains some complete or incomplete hernias that are not found on physical examination, although they may be demonstrated several days later.

The relation of the sac to the cord is important. If it is posterior to the cord, it indicates a congenital affair, since the testicle and tunica vaginalis descend posterior to the cord, leaving any persistent processus vaginalis in that location.

The degree of herniation permits a relative differential classification into: (1) Incomplete hernia, in which the smooth sac, which gives an impulse on coughing or straining, is palpable in the external ring, but does not form a hemispherical bulge; and (2) complete hernia, indicated by the same general condition, but in which the sac forms an arc of 180 degrees or more.

Fluid in the sac may be divided into: (1) Clear fluid, which is a normal content, since all closed cavities supporting vital structures have a fluid secretion; or (2) bloody fluid or other abnormal content, indicating either a pathologic condition in the abdominal organs draining into the sac, or trauma to the cord by careless operative manipulations. (Taruma to tissues in the passage of a hernia would produce blood external to the sac, which would be found as clots in the canal.)

Hernia produced by digital examination would react the same as an acutely acquired hernia, since the same structures would be trauma-

tized and an activ ehernia would result. Complete hernia may be produced by digital manipulations traumatizing the supporting structures of an incomplete or potential inguinal hernia. The pain would be proportional to the number of structures injured and to the extent of the injury.

It is the duty of surgeons to clear up any controversy in this field by keeping operative data, showing that the industrial attitude is or is not well founded, if this subject is approached from a congenital predisposition angle.

Injection Treatment of Hernia

THE object of this method of treatment of hernia is to produce an artificial proliferation of fibrous tissues without suppuration, which will close permanently the defect in the abdominal wall through which the hernia protrudes.*

For more than one hundred years attempts have been made to accomplish this object, but unfortunately many of the efforts have been made by men unfamiliar with anatomy and asepsis, and by quacks who employed such foreign bodies as high melting point paraffin to obstruct the aperture. Velpeau of Paris as early as 1835 used irritants with apparent success to cure hernia. Heated instruments have been used to cauterize the surface of the canal in inguinal hernia, in the attempt to obliterate the defect by scarring and subsequent constriction. Extremely irritating solutions have been injected carelessly into the abdominal wall near the hernia, sometimes without the patient's even wearing a truss. Disastrous results have followed, causing the medical profession to look with a jaundiced eye upon any endeavor to treat a hernia by injection, and the public has had good reason to fear the many complications which have occurred, such as peritonitis, fecal fistula, gas gangrene. and even death. To this may be added the statement that many unethical men have sold and are now selling worthless solutions and making extravagant claims for their value, thus further prejudicing the medical profession against this treatment. Although some criticism has been leveled at this treatment because of the claim that it is impossible to blindly inject this area accurately, it has been found that in cases in which methylene blue is injected. and the patient subsequently operated upon, the methylene blue has been deposited quite accurately in the area for which it was intended. Another interesting observation is that when the external ring is injected the methylene blue is seen to travel along the lymphatics to the internal ring, thus spreading through the entire area of the inguinal canal.

About three years ago I first became interested in this study, and observing the excellent work of Drs. Bratrud, McKinley of Minneapolis and others, I decided to investigate this method. Following the successful use of the method over a period of a year the opportunity was afforded to made a study of it at Northwestern University Medical School both in the experimental laboratory and clinically. By the use of experimental animals the results were studied following the injection of various irritants at different locations. Several solutions were discarded as producing no reaction or too severe a reaction, and two solutions with the following contents were selected to be used clinically:

Thuja Solution containing Phenol 30 c. c.

Grain Alcohol 15—Lloyds specific Tincture of Thuja 15.

I. Tannic Acid solution.

This solution is used in three different strengths.

It is a distillate solution prepared by distillation of the tinctures of a group of tannic acid bearing herbs to which the following ingredients are added:

Benzyl Alcohol — 3%; Thymol — 0.5%.

Reagent grade natural tannic acid is then added to make the resultant solutions assay "A" solution—¼% tannic acid, "B" solution—½% tannic acid and "C" solution—1% tannic acid.

These solutions were injected in doses sometimes two or three times that to be used in the human and the injections were made at the following sites; the blood stream—directly into the abdominal cavity—the spermatic cord — the testicle — and the fascia about the inguinal canal. There were no fatal reactions and aside from transient respiratory and blood pressure variations the only reactions noted were tissue changes without necrosis, except those animals in which these solutions were injected into the abdominal cavity revealed on opening 10 days later a discrete area of necrosis with beginning healing. The microscopic changes so far as those occurring in the fascia about the inguinal canal will be described later in detail by Dr. Cunningham, who aided me in this work. I would, however, like to say here that there was no tissue necrosis and there was new fibrous tissue production which went on to form firm permanent tissue with only partial absorption.

No atrophy of the testicle was noted either when the solutions were injected into the spermatic cord or directly into the testicle although here as in the fascia inevitable changes took place as will be described later.

In selecting cases for treatment the following conditions were considered to be contra-indications: sliding hernias, incarcerated irreducible hernia, hernias associated with undescended testicles, hernias where the defect was obviously too large, diseases producing ascites, markedly enlarged prostate, chronic severe coughs, hemophilia, syphilis and toxic goiter. I might add that patients who were not relieved of all symptoms by wearing a truss as well as neurotics were not treated by this method. All patients were carefully examined for any sign of strophy of either testicle before treatment was instituted. It is surprising the number of patients presenting themselves for relief of hernia who have such atrophy, our experience showing about 8%.

Considerable trouble was encountered at first with trusses but generally we found that in selecting a truss the pressure must be applied anteroposteriorly and not circular—a special spring type with a non-skid pad is therefore the type we are using. This truss must hold the hernia reduced at its point of exit at all times, and should be fitted sufficiently firmly to produce an actual depression in the skin without damage thereto. It is at once apparent that with an indirect inguinal hernia the pad must be placed high over the internal ring, and with a direct inguinal hernia it is

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*W. M. McMILLAN, M.D.; read before the Section on Surgery, 86th Annual Meeting, Illinois State Medical Society, Springfield, May 20, 1936; and published in *Illinois Medical Journal*, September, 1936.

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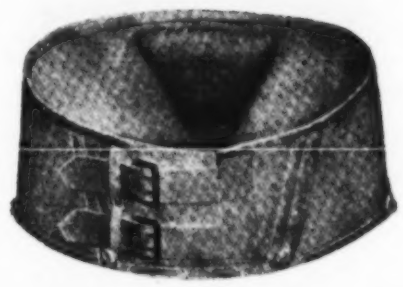

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placed lower over the external ring. Failure to observe these details and application of a proper truss will result in inability to cure the hernia. (I might say that the fitting of trusses now is largely in the hands of drug store clerks.) The truss must beworn day and night for one one to two weeks before treatment is started. The purpose of this is to prevent interference with the treatment after it is once instituted due to skin irritation, and most important of all to be sure the hernia is reduced and can be continuously held so. After treatment has been carried on for one to two months the truss may be removed at night after retiring and applied again in the morning before arising. The truss should not be removed permanently until one is reasonably sure that the hernia is firmly closed, which is usually found to be two to four months from the time of beginning treatment. In the case of large hernias it is necessary to have the patient wear the truss day and night for a longer period. At all times when the truss is being worn it must be removed and replaced with the patient in the supine position.

The patient must be instructed that although he may carry on his normal activities while under treatment, the pad must at all times be in proper position, and if necessary a perineal strap must be worn.

As stated previously the thuja mixture and the tannic acid, alcohol solutions were because of their satisfactory action chosen for our clinical work. Other solutions were not used because they gave no local reaction or too severe a reaction or resulted in a general reaction simulating shock.

Dosage. In the case of thuja solution four to eight drops was the dosage used; and of the tannic acid solution one to four cubic centimeters, depending upon the strength. When the latter solution was employed, a preliminary injection of one cubic centimeter of two per cent. novocaine solution was required. After its injection the needle was left in situ for a few minutes, and the tannic acid solution was then injected. The minimum dose was used in the region of the internal ring and the larger doses along the canal and the external ring. A tuberculin syringe was used for the the first solution, and a five-5 c.c. syringe for the latter. A one and one-half to two-inch 22 gauge needle was used, preferably the security anesthesia type to prevent the possible accident of breaking the needle within the abdomen.

Before treating the patient the following general rules must be laid down. One must be certain that the hernia is reduced. The patient must be placed in the supine position and the truss removed and replayed by the surgeon, unassisted by the patient. The skin is prepared by washing with 40% alcohol. The solution is injected slowly and firm pressure applied to the site of the injection for a few minutes thereafter. As the majority of our cases were of the inguinal type the technique for this type of hernia will be given in detail. The internal ring is located slightly above the mid-point on a line drawn between the anterior superior spine and the symphysis. These three points represent the landmarks which are used as a guide in the treatment of this type of hernia. The direction of the needle should be slightly caudal and not directly vertical to the abdomen. All cases should be injected along the entire course of the canal regardless of the type of hernia. One should first inject the area through which the hernia protrudes, and subsequently the remainder of the canal. In addition, some of the solution must be placed in Hesselbach's triangle, depositing the solution along the conjoined tendon and upon the transversalis fascia. In this case the needle is introduced mesial and posterior to the cord. A distinct "give" sensation is experienced as one goes through the fascia. The syringe must be aspirated before the injection is made to be sure the needle is not in a blood vessel. When injecting the solution about the external ring, it is obviously easier accomplished under the direct guidance of the surgeon's finger which is inserted into the ring by invaginating the scrotum. When injecting the internal ring you are at once aware of the fact that the point of your needle is therein because it can be moved freely in any direction. In most cases the patient will complain of a slight sensation of pain along the cord and in the testicle on the side of injection.

In the case of femoral hernia the solution is used in the minimum dosage and the needle must be placed very carefully in the femoral canal medial to the finger which is placed therein, in order to avoid injuring the femoral vessels and nerve.

In incisional hernia the minimum dosage is also employed, and extreme care must be exerted to deposit the solution directly into the fascia without injury to a viscus. The same obtains in the treatment of umbilical hernia.

The number of injections vary according to the type of case, the average being eight to twelve, although sometimes as few as four or five and as many as twenty-four have been necessary. As few as four are not recommended as the tissues should be over-built to allow for absorption. The average case is injected once or twice weekly depending upon the reaction. The patient should not be discharged until the entire area through which the hernia has presented itself is firm and no impulse can be made out on coughing. The patient is advised, on being finally discharged, to return for a check-up twice yearly, to avoid constipation and to reapply the truss if seized with any unusual and prolonged coughing spells.

As for complications, we have to date not observed any more serious than a temporary swollen, painful condition of the cord, which was relieved by hot or cold applications, and no patient has been entirely incapacitated for more than one or two days. To date, we have not observed a hydrocele of the cord which required aspiration. They have all subsided within a week or ten days without any treatment other than local applications.

Three reactions have to date occurred where the patients complained of some abdominal pain and have exhibited marked weakness; breaking out in a cold sweat and simulating a mild degree of shock. All symptoms disappeared in an hour without treatment except in one case which was removed to a hospital because of the relatively severe symptoms. He made an uneventful recovery and left the hospital a day later without special treatment. This case I am inclined to believe can be explained on an allergic basis. So far as impotency or sterility is concerned, this has not to date been accurately checked, but we have had not complaints or reports of this in any of our cases.

In summarizing my experience with this method I would say that it covers about 400 cases treated, of which about 200 have been discharged as cured. The recurrent rate is to date 8%. My oldest case from a standpoint of date discharge is three years. There have been eight cases where a closure could not be obtained. The selection of cases was very liberal, excluding only the cases showing definite contraindications. Many of them were surgical recurrences. Contraindications were found in about 10% of the cases examined. The age range was 2 to 90 years.

In conclusion, the author believes that this procedure should be regarded as a surgical one for obvious reasons and if it has merit—as our experience and that of others indicates—it should be placed in the hands of the regular surgical profession where it belongs. To date we can say that this method is as safe or safer than surgery, the incidence of recurrence is less than after surgery and where recurrences do result they are invariably smaller than the original. In addition, the further procedure necessary for a cure is relatively simple as compared with a new operation such as is required after surgical recurrence.

It might be stated that it is too early to be sure of our percentage of recurrences but unfortunately the same can be said of the surgical recurrences as there is a wide difference of opinion regarding it. We do feel that our figure is quite accurate as we have adop'ed a careful follow-up system and the patients seem very willing and anxious to return for further treatment, which cannot be said of many of the surgical recurrences.

In addition the economic side of this method of handling hernia must not be lost sight of as the hospital expense and loss of income from absence from duty for several weeks is spared these patients.

The future may show the advisability of more careful selection of cases. We feel the field is open for further investigative work carefully controlled on the basis of our present results.

Finally we must not forget that accidents can and will happen if those attempting this procedure are unfamiliar with it or careless in carrying it out.

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Discussion

DR. D. R. CUNNINGHAM, Evanston, Ill.: I am going to deviate somewhat from the regulation rôle of a discussant to Dr. McMillan's very splendid paper, because I was associated with him in a great deal of the work that he has here presented.

Dr. McMillan has mentioned that opportunity was afforded us to make a study of the results of injecting irritants into the tissues of dogs. I would like, at this time, to show a few lantern slides of microphotographs of tissues to demonstrate exactly what does take place after sclerosing irritants are introduced into the inguinal canals of dogs. The first slide shows proliferating fibroblasts and remnants of lymphocytic infiltration, in a section of tissue removed from the inguinal canal of a dog, one week after the latter was injected with thuja mixture. The second slide shows granulation tissue and maturing fibrous scar two weeks later from the same area as slide number one. The next slide shows evidence of maturing fibrocytes and remnants of lymphocytic infiltration, and the last slide shows mature connective tissue together with old scarring in a section of tissue eight weeks after the first injection was made. My purpose in showing these slides is merely to demonstrate that the theory of initiating new firm fibrous tissue in the inguinal canal to obliterate a defect is not without foundation. However, I am of the opinion that the biggest disappointment that will befall the operator who uses this method is this; after the entire inguinal canal has been injected in the course of several treatments and no impulse can be found either at the external ring or at any place along the canal, the surgeon will, after the lapse of some weeks, note that a good bit of his newly formed fibrous tissue has been absorbed and that the hernia has recurred. In other words, how can this scar tissue be made permanent? I do not attribute much worth to the claims of surgeons using this method or to the manufacturers of certain solutions for this purpose, that one solution has superior qualities over another in the production of fibrous tissue. When a reaction occurs from whatever solution is used the same final result is lymphocytic infilteration, new fibroblasts, and finally mature connective tissue. Dr. McMillan has mentioned the necessity of a properly fitted truss. It is quite surprising to see a patient come to the office wearing a truss, which is absolutely useless to him. The mere fact that some pressure is exerted at some place along the inguinal canal, very likely explains why some of these patients feel fairly well even when wearing an ill-fitted truss. When the truss is applied properly and has proven to retain the hernia, in any position the patient may assume, all symptoms of discomfort and "heaviness" and "drawing" in the inguinal area are promptly relieved. Dr. McMillan has mentioned that one must be sure that the hernia is reduced. Very often small hernias are not detected and the contents could, more often than not, be injected if not recognized as unreduced. I am of the opinion that the cases cited by Dr. Richard Goldhahn must have been complicated by abdominal viscera in the sac. Goldhahn reported three cases coming under his own observation, consisting of two cases of fecal abscess and fistulae and one case of gas bacillus sepsis, and cited two other of much the same complication. I mention this because Dr. McMillan's paper may leave the impression that this technic is quite simple. I think that any needle one and one-half inches long placed in the inguinal canal, first assuming that the needle is there, and then introducing phenol or tannic acid or some other irritant entails some amount of risk. I think the method is safe enough in the hands of men who have access to a large number of cases for treatment and enjoy a good deal of experience.

When the method becomes more popular, as it is doing constantly, a great many accidents are bound to happen. Whether or not the method will live as a result of what will inevitably take place by the inexperienced operator in the future, is a matter of speculation. We do know that the number of recurrences in the surgical management of hernia has dampened the enthusiasm of the public, somewhat, in this respect. Surgical intervention today for all types of hernia, is not entirely satisfactory. While the Bassini operation or same modification thereof, may give a fine record of successes in the hands of good surgeons, nevertheless, there still remains that group to which the same operation becomes a repetition. In many cases patients are operated upon on the basis of symptoms only, or because the examiner has found an "impulse" with his finger in the external ring. Many of these patients have a slight bulging suggestive of a direct hernia, where there is no indication for surgery. On the contrary, there are many patients who complain of a certain amount of discomfort in the area of the internal rings, on one or both sides. These patients have distention and gastrointestinal symptoms and are often diagnosed as colitis, etc. In many of these patients, one cannot definitely diagnose an indirect hernia because the neck of the sac is kept fairly well closed by the muscles at that point but as a weakness develops, inflammatory changes occur about the neck of the sac as it tries to progress forward and the resulting symptoms, referred to to peritoneum, occur. I believe in these cases the injection treatment with some support at the ring is ideal treatment. Personally I do not think we are justified in operating on a patient where we cannot definitely convince ourselves that a hernia exists.

The late William B. Coley reporting on 100 cases treated by injection, gave us the sad story of 100% recurrence. However, these figures differ widely from those reported by others who are using the injection method. I think it is quite interesting to think about the work of Dr. McBurney, some 40 years ago, who after widely incising the external aponeurosis, and removing the sac high beyond the internal ring, packed the open wound with iodoform gauze to induce scarring of the whole area. It was soon noticed that what was firm connective tissue at one time, gradually became thinner and recurrences resulted. Much the same type of scarring is anticipated with the injection method except, unlike McBurney, the fascial planes are not disturbed. I think it is well that we present the method open-mindedly, in view of the doubts of efficacy of the method, we have entertained in the past. Dr. McMillan has presented a resumé of the treatment in a purely investigative manner, and leaves us to judge for ourselves, as to its usefulness. If

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the method is not satisfactory, it will not be long before it becomes discarded, and if it has merit it will be kept and used by those who can satisfy themselves with its results.

DR. GEORGE DE TARNOWSKY, Chicago: It is interesting to go back 50 or 75 years to the injection treatment of hernia. I feel that as a surgeon Dr. McMillan's paper rather justified our results. When we speak of failures in herniotomy we include not only the oblique, but the direct and the ventral hernia. McMillan's method has very limited application. He mentioned the minor complications, but he forgot to mention direct hernia. Any large hernia is not suitable for injection treatment. The sliding hernia is not suitable. It is quite true that Dr. McMillan and his associates have taken up this study scientifically by experiments on dogs before trying it on the human being. It is true that we have a great number of sclerosing agents, and it is true that scar tissue will thin out. Dr McMillan obtained his best results in the small, oblique hernias, but those the the hernias in which we get 100% good results with surgery.

He spoke of the economic saving. The patients have to come back for check-up of these hernias repeatedly. When you analyze your entire treatment from an economic viewpoint, I do not think there is much to be gained over the knife of surgery.

I would question its use in femoral hernia. There is not much in femoral hernia except the sac, only in rare cases where it passes above Poupart's ligament and makes the surgeon think he is dealing with a direct hernia.

I think the word of warning brought out is worth repeating. If any of you are going to attempt the injection treatment, try it out very carefully on the cadaver. Get the anatomic points carefully outlined, and then watch for the complications. If this becomes popularized, that is, taken up by the casual operator, I fear we will have as many calamities as we read about fifty or sixty years ago.

DR. C. E. WARD, Cleveland, Ohio: It has been a great pleasure to know Dr. McMillan and to have had the opportunity of attending his clinic at Northwestern University and I have been very much impressed with the results he has had with the injection treatment of hernia.

During the past 36 years I have operated on several hundred hernias and my results have been about the run of the mine, the average number of cures and recurrences. I did not accept the injection treatment of hernia overnight but made a thorough study of it over a period of several years before adopting this method of treatment. I believe it offers a tremendous field for any man who will study his anatomy minutely. Personally, I went back to my cadavar work and injected methylene blue into the inguinal canal and observed where it flowed. This gave me definite knowledge of the channel the fluid follows when injected into a patient.

No one should attempt to inject a hernia unless he is definitely sure that it is completely reduced and can be held reduced by a truss. After you are satisfied on this point the most important step then is to be able to differentiate the types of hernia. We all should be able to do this but, unfortunately, we all cannot. Mistaken diagnosis as to the type of hernia has caused much of the failure and criticism of this method of treatment.

It is most essential that you know how to reduce a hernia, and to know when a truss is well fitted. Dr McMillan has mentioned the importance of the truss and I thoroughly agree that while it is not within the province of our profession to fit the truss, we should know when it is well fitted and should have one truss man upon whom we can rely. When I send a patient to a truss maker, I send the diagnosis and tell him how the truss is to be fitted. If you will watch your truss, you will eliminate a great deal of trouble.

Concerning contraindications, I think Dr. McMillan has been overly cautious. I believe that none of us would attempt to operate on a hernia on a man 76 years of age with a large simple hernia, complicated with either nephritis or diabetes or an enlarged prostate and one in whom the hernia rolls out every time he attempts to void but I can take that same patient into my office and, if I can reduce his hernia and hold it reduced with a truss, I feel absolutely confident that I can give him more relief by injections than I would by surgical procedure, and this without danger to his life. When we meet with an elderly gentleman with a hernia which a truss will not hold, we can put him to bed for four or five days, give him three or four injections which will create a sufficient amount of exudate and fibroblasts so that his truss will hold the hernia reduced and then continue treatments as an ambulant case.

I was surprised to hear Dr. McMillan say that he had had one gentleman 90 years old. My oldest patient is 76 years of age and my youngest one year. I find that about 95% of the cases which I have seen are male patients and 5% females. They are subdivided into four groups, direct, indirect, femoral and postoperative hernias. Of 100 hernias, you will find 10 recurrences, postoperative patients coming for relief who have been operated on as many as three times. Many have had fascial transplants.

Twenty per cent of the hernias are direct, that is my average. Some statistics show it to be lower. Bilaterals will run about 10%. Right hernias occur twice as often as left hernias.

The Industrial Commission of Ohio at the present time recognizes my work and allows me to treat patients by the injection method. I do not know whether or not your Illinois Commission recognizes it.

As to the preparation used. I feel that we will, in all probability, have a better preparation than either tannic acid or thuja mixture. I firmly believe that no one mixture will cure all types of hernias.

I treat my cases individually and sometimes use a combination of thuja and tannic acid. My practice is a referred one and has to be carefully handled, and to minimize the discomfort, I have worked out what I call a "Triple A" anesthesia. I first apply to the skin where I am to make my injection a few drops of a solution of a 10% benzocain dissolved in one dram of alcohol to which is added one dram of 5% carbolized oil and one dram of castor oil. The castor oil is used to hold the solution in the spot where you are to make your injection.

This solution acts as a local anesthetic to the skin and undeniably lessens the pain when the needle is inserted. The second anesthetic is a solution of novocain and diothane. The diothane is similar to novocain but more prolonged in action, lasting, as a rule, for several days. I use 2 cc. novocain with ½ cc. diothane. I use first a very fine needle and make a small wheel into the subcutaneous tissue, then use a 1½ inch, 22 gauge needle and inject the remainder of the novocain mixture into the canal, leaving the needle in situ. About five minutes later I slowly inject the sclerosing fluid through this same needle into the inguinal canal. When you feel the needle pierce through the fascia you will know you are in the canal. For my convenience and to avoid any confusion between the two solutions, I use a blue syringe for the novocain and a white one for the sclerosing fluid. I find it essential to keep a record of each injection on a card which I have had printed for this purpose.

Under complications, Dr. McMillan has said that he has never had a hydrocele of the cord following injections. I have had three following direct hernias. From a surgical standpoint direct hernias are difficult to operate on and the results are not as satisfactory as the other types and more frequently recur. Direct hernias are also more difficult to treat by the injection method and recurrences are most frequent.

In injecting the fluid in to Hesselbach's triangle, one may get a few drops of the fluid into the upper or lower angle of the cord and in this event a hydrocele might result and if it does, aspiration may be necessary. One patient showed an allergy to tannic acid. He had bilateral hernia and I treated both sides at the same time and I feel sure that I gave him too large a dosage. The ill effects passed away in about three hours, however. I have also seen swelling of the testicles as the result of a treatment. A suspensory was applied and they were made comfortable and the swelling quickly subsided.

One point I want to bring out especially: I have not seen an infection or a necrosis. If you know where your needle is, certainly this treatment is just as safe as any surgical procedure that has generally been adopted.

DR. JOHN A. WOLFER, Chicago: I want to take this opportunity, as this work comes from my school, to express the policy in our division. Three years ago at a meeting of the American Surgical Association in Toronto, Dr. Wangensteen, Professor of Surgery at the University of Minnesota, told me that if he had a hernia he would have it injected and not operated upon. Knowing him as I do, I thought there must be something to it. It is the duty of a Uni-

THE BULLETIN

of

The American Association of Industrial Physicians and Surgeons

THE object of this Association shall be to foster the study and discussion of the problems peculiar to the practice of industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes and results of the medical care of employees, and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for the mutual advancement in the practice of their profession.

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versity to find out whether a suggested procedure has real merit. There is no other way we can teach or advocate for or against a procedure unless it is carefully investigated, therefore, Dr. McMillan started to work out this plan. They understood the situation perfectly well and went into this with an open mind. They are going to study their cases carefully. The basic principles have been studied in experimental animals. I want to express this as the policy of our Department of Surgery. The reason for the frequent visits was for the purpose of observation and does not mean that the patient who has received the injection method of treatment need to return so frequently.

DR. W. M. McMILLAN, Chicago sing): I have nothing further to say except to clear up one point. We did not eliminate direct hernias in our series. We do not consider them contra-indications, though I know they are more difficult to treat and cure. Our recurrence rate includes direct hernia.

The Inguinal Trigone: Industrial Considerations

THE purpose of this paper is to suggest a different conception of industrial hernias and to propose means whereby, through the guidance of the medical profession, responsibility may be assumed by industry in all hernias occurring during employment* — a responsibility sufficient to relieve the disability of the workman at a cost not prohibitive to industry.

Personally, I believe that: The medical care of injured workmen, which plays such an important part in the administration of workmen's compensation, should be considered a direct responsibility of the medical profession; organized medicine through state and county societies should so direct and supervise the practice of industrial surgery that the disability of the injured workman and its relief through adequate medical care may ever be the first thought in such practice; and that organized medicine should hold itself responsible to the employer for adequate medical treatment at a cost consistent with the services rendered and the ability of industry to pay.

Definition and boundaries: The inguinal trigone, or inguino-abdominal region, as described by Callander, is that subsidiary area of the anterior abdominal wall on either side, bounded below by the inguinal ligament, above by a horizontal line extended from the anterior superior spine of the ileum to the lateral margin of the rectus muscle, and mesially by the lateral margin of this muscle.

As the surgical importance of this region is derived from the high incidence of inguinal hernia, so is its industral importance of moment because of the great number of inguinal hernias developing in workmen during employment, and further, because of the responsibility which industry must assume for the disabilities resulting therefrom, more especially in the light of the principles of workmen's compensation and the laws enacted for the government of those principles.

The workmen's compensation laws, enacted by the various states of the Union, contain one or more sections dealing specifically with hernias. In all of them, so far as I am informed, wil be found definite restrictions excluding all hernias except those which are described as real traumatic hernias.

The Arizona Law, section 1439, reads as follows:

"All hernias are considered to be injuries within the provisions hereof causing incapacitating conditions or permanent disability, and until otherwise ordered by the Commission, the following rules for rating the same shall govern: (a) Real traumatic hernia is an injury to the abdominal wall of sufficient severity to puncture or tear asunder the wall, and permit the exposure or protruding of the abdominal viscera or some part thereof. Such injury will be compensated as a temporary total disability, and as a partial permanent disability, depending upon the lessening of the injured individual's earning capacity; (b) all other hernias, whenever occurring or discovered and whatsoever the cause, except as under (a), are considered to be diseases causing incapacitating conditions, or permanent partial disability; but the permanent, partial disability and the causes of such are considered to be as shown by medical facts to have either existed from birth, to have been years in formation, or both, and are not compensatory, except it be proven: That the immediate cause, which calls atten-

* R. F. PALMER, M.D., Medical Adviser, Industrial Commission of Arizona, Phoenix, Arizona; presented at the 45th Annual Session of the Arizona State Medical Association, April 23-25, 1936; published in *Southwestern Medicine*, June, 1936.

tion to the presence of the hernia, was a sudden effort or severe strain or blow received while in the course of employment; that the descent of the hernia occurred immediately following the cause; that the cause was accompanied, or immediately followed, by severe pain in the hernial region, and that the above facts were of such severity that the same were noticed by the claimant and communicated immediately to one or more persons, in which event they are considered to be aggravations of previous ailments or diseases, and will be compensated as such for time lost only to a limited extent, depending upon the nature of the proof submitted and the result of the local medical examination, but not to exceed two months."

Under a strict interpretation of this law and the rules now governing its administration, a traumatic hernia is seldom encountered and then most probably only in association with other serious injury. Likewise, a compensable hernia, if fully meeting the requirements laid down for it, is unusual. All other hernias, and they comprise more than 95% of those reported to the industrial commission of this state, are entirely unprovided for.

Hernia decisions by the Commission: When a hernia occurs in a workman, he, either then or sometime later, files claim for compensation. The industrial commission, in order to act on the claim and determine the industrial responsibility must, in addition to the workman's statements, have the employer's report of the accident and the initial medical report of the physician who examined the patient. If these two reports and the workman's statements do not agree as to details of the accident and subsequent events, and they frequently do not, an investigator is put on the case to acquire the possible facts. An examination by a second surgeon or the medical advisor frequently is requested, and, as a rule, considerable time and expense are involved before sufficient facts are available to warrant a decision. Should responsibility of the insurance carrier be denied by the commission, the claimant, who firmly believes the hernia was caused by the strain, demands a formal hearing, and he starts looking for doctors, lawyers and political friends seeking assistance in securing the consideration to which he feels himself rightfully entitled. Consequently, further waste of time and money is incurred before thought can be given to the physical ailment of the man and the means by which his disability may be relieved. It might appear that if the doctors who first see the hernia cases would make thorough investigations and reports and carefully explain to the patients just what a hernia is and why the commission cannot accept responsibility, or place it upon the insurance carrier involved, or order operation and compensation, many of the difficulties surrounding these cases might be solved. This, however, is too much to expect even from a member of the medical profession, at least, under the present industrial set-up. As a matter of fact, a composite of fully 90% of the initial medical reports coming in on hernia would read somewhat as follows: "While on duty carrying a heavy object, patient noticed a pain in the right groin. Examination made later, reveals a tender relaxed inguinal ring with impulse on coughing. Diagnosis: traumatic inguinal hernia. Treatment: hernia reduced; patient ordered to keep off his feet. Prognosis: total disability until operated." Under remarks will be found: "Request permission to operate." While the man doubtedly has a hernia, such a medical report is hardly consistent with other facts surrounding the case and is obviously of little value to the commission in determining cause and effect and in deciding responsibility under the written law. While a more accurate medical report might clarify the situation, nevertheless, it would not remedy the basic faults which lead to so much misunderstanding and indecision regarding industrial hernias.

Underlying faults in industrial conception of hernia: In the legal and lay conception of hernia, as expressed in the workmen's compensation law and the rules provided to administer it, the underlying thoughts deal entirely with the character of the accident and the time effects of the injury. While this conception usually leaves no doubt in the case of "real traumatic hernia," where the accident has been severe and the results definite, it leads to great confusion and dissatisfaction in those described in the law as "all other hernias." In the other than real traumatic cases, it very occasionally happens that the strain or blow was such as to cause an immediate descent of the hernia, accompanied by severe pain, and that the claimant immediately communicated such facts to one or more persons. As a rule, however, one or more of these various immediate elements is lacking and the cases gradually shade off from the conditions required to make a hernia compensable, to the strain where the only symptom is a local discomfort of mild degree and the hernia remains incomplete and only potential. Manifestly, the variations in these hernias are of degree only. All of the other than real traumatic hernias are preformed and potential for varying periods, and when conditions are right, no matter how severe or mild the strains, or whether the hernias protrude through the rings all at once or gradually over days or weeks, the ultimate effect on the man is the same. It therefore appears that if in any of these other than real traumatic hernias, industrial responsibility is to be assumed, it should be assumed in all, for even the least of these may cause the greatest disability; and the relief of disability is the foremost principle behind workmen's compensation and of the industrial surgeon.

Can we not reclassify hernias from a clinical and disability standpoint and co-ordinate this with industrial responsibility and provide ways and means of relieving disability in all cases of industrial hernia. I would place all hernias in two groups.

In group one are the real traumatic hernia, as defined, and the strangulated hernia, both emergency surgical conditions, and the incompletely reducible hernia, not necessarily an emergency condition, nevertheless, disabling and only relieved by operation. The clinical indications are clear and the responsibility of industry is only limited by other than medical considerations surrounding the case. These cases should have both treatment and compensation.

In group two are placed all other hernias of whatever degree or cause occurring during employment—more than 95% of all industrial hernias. In practically all the immediate disability is moderate and relieved by the application of a properly fitted truss. This much, at least, industry should be responsible for; it is, therefore, suggested that responsibility for treatment shall be immediately accepted in all group two cases, no matter how trivial the accident may appear, and that the attending surgeon shall be authorized to provide the workman immediately with a truss. The attending surgeon shall make a full and comprehensive report to the commission on a special blank for that purpose. For this service examination and report, the attending surgeon shall receive a suitable fee. There will naturally be a number in whom permanent wearing of trusses will not be satisfactory. The immediate disability, the first and often the only responsibility of industry, has been relieved, without loss of time for the workman or undue expense to industry. Further consideration of each case may then be given with additional procedure as may be deemed expedient and proper by the commission.

The experiences of the Arizona Industrial Commission in relation to the industrial hernia problem has probably not been vastly different from those of similar bodies in other states. Up to perhaps 1927 or 1928, comparatively few hernia claims were accepted for compensation and operation. As time went on, however, the commission, often under pressure, and with no sharply defined line to be drawn between the compensable and the non-compensable cases, gradually extended medical and compensation benefits 'till by 1933, when the present administration took office, a large proportion of the cases were being accepted for operation or compensation or both.

The costs of investigation, treatment and compensation increased tremendously, not only in hernia cases, but in many other conditions, and this, together with greatly lessened premium payments, was seriously menacing the compensation fund. To meet the situation, the commission, along with other economies, attempted to reduce hernia costs by adhering strictly to the stipulations in the law. For perhaps a year, this policy was successful, at least as far as compensation and medical costs were concerned. The disability of the workman, however, was receiving scant consideration, and because of exaggerated reports, demand for consideration by employers, employees and lawyers, and because of the illy defined provisions in the law, constant argument, expensive investigations and economic losses obtained. Consequently history repeated and the commission now finds itself confronted with the problem of again readjusting its attitude in dealing with hernia cases.

It is to meet this situation that I ask the medical profession to discuss the problem and to assume its natural

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responsibility by leading the way.

As a comparison of costs and to indicate what could be done under the suggested manner of treating industrial hernias, I have taken the files of 1934 as representing an average year. During 1934 there were 111 hernias ranging from inguinal strains with incomplete hernia to complete hernias.

Of the 111 cases, 63, or 56.75% were awarded compensation and operations at an average cost of $325.00 per case—a total of 20,462.00 dollars.

Forty-eight, or 43¼% were compensated to the extent of actual time lost and medical costs, an average of $28.00 per case— a total of $1,352.89.

The total for the 111 cases was $21,814.89, an average of $196.53 per case. These costs include medical, hospital and direct compensation only. They do not include costs of investigation, time lost by workmen in pressing claims and numerous other economic and monetary losses. Neither do the 111 cases represent all of the hernias which developed during the year for many were not reported. The number reported in 1935 is probably double that for 1934, and at the present time claims are coming in almost daily. For purposes of discussion and comparison, however, the group is sufficient.

Assuming that 5% of the 111 cases would come under group one of the proposed classification and that the average cost of $325 per case would continue, and assuming further that the cost of examination and report, plus fitting truss would be $15.00, and assuming further that 50% of group two cases would be later treated by subcutaneous injection at an average cost of $50.00, we have the following:

Group				
One ..	5½ cases	@	$325.00	$1,787.50
Two	105½ "	@	15.00	1,582.50
Three	52¾ "	@	50.00	2,637.50

An average for the 111 cases is $54.12 per case.

It is possible that some cases in group two may require compensation for short periods and that some of them surgery and perhaps a greater proportion will require injections. Such additional cost could not exceed two-thirds of the total and there would still be a cost saving of more than 50 per cent, together with the elimination in great part of all argument, excess investigation costs and various other economic and monetary losses. Even a greater consideration perhaps would lie in the fact that industry would be assuming its rightful responsibility to employees and all hernias occurring in the course of employment would be adequately provided for.

I am not taking up technique or relative merits of the three forms of treatment, retention by truss, truss plus injection and cure by operation. Suffice it merely to mention that there is ample recorded experience and evidence to state that:

1. Disability in a great majority of hernias may be satisfactorily relieved by properly fitted trusses;

2. Approximately 98 per cent of hernias treated by injections are completely closed and permanently retained and, in competent hands is free from discomfort or complications and does not incur great expense or loss of time; and

3. The cure of hernia by operation is necessary in a small percentage of industrial hernias.

CONCLUSIONS: 1. Hernia is of frequent occurrence in industry and is a serious industrial problem.

2. Except in conditions described as "real traumatic hernia," the present workmen's compensation laws and the rules which govern their administration lead to much confusion, dissatisfaction and unnecessary expense in handling industrial hernias in Arizona.

3. In hernias classified in the law as "all other hernias," the distinction between the one termed compensable and the one termed non-compensable is of degree only.

4. Relief of disability is one of the underlying principles of workmen's compensation and should be the first thought in medical treatment.

5. If industry as a whole is responsible for any "other than real traumatic hernia," it should be responsible for all, at least, to the extent of relieving the immediate disability.

6. In the vast majority of industrial hernias, the immediate disability can be relieved by the application of a properly fitted truss at small cost to industry and without lost time for the workman.

7. A plan is proposed whereby through a clinical and disability classification of industrial hernias, all cases can be immediately accepted as responsibilities of industry and appropriate treatment inaugurated without expensive delay.

8. The savings in operative fees, hospital costs, compensation and investigation costs and numerous economic and monetary losses will permit industry to accept its rightful responsibility in all industrial hernias.

9. A considerable study of the subject has convinced me that the suggested procedure is practical and sound and that with the leadership and cooperation of the medical profession of the state, it can be successfully carried out and result in a satisfactory solution of the industrial hernia problem.

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Treatment of Hernia by the Injection Method

IF TOLD that injecting a mildly irritating solution into a canal approximately four cm. long produces in from 30 to 40 days a dense plug of fibrous tissue obliterating that canal and fusing the surrounding structures, you would probably say, as many surgeons have said, "it can't be done."* Shown, microscopic sections of strong dense connective tissue produced by such injections in animals and in humans and, thousands of patients who formerly had inguinal hernia and now have their inguinal canals obliterated by connective tissue from such injections no evidence of hernias, would you still say, "it can't be done?"

Professor Billroth, the great German surgeon, had the vision to predict that if a solution could be perfected which, injected into the inguinal canal, would cause proliferation or connective tissue, operation for cure of hernia would be practically eliminated.

For the past 100 years attempts have been made to cure hernia by introducing into the inguinal canal substances to produce chemical inflamation and scar tissue to close the canal. The chemicals formerly used were often so powerful that abcesses, destruction of tissue, peritonitis, and even fatalities resulted. The ethics of the gentlemen doing such work were often questionable, and they justly received the condemnation of the medical profession. No animal experiments were done to find non-toxic, non-caustic, solutions. No histological examinations were made to prove that normal connective tissue was produced.

During the past few years interest has been revived in the cure of hernia by injections by men whose standing is unquestioned. Much experimental work has been done on animals to prove that the solutions used are non-toxic in even the peritoneal cavity and veins. The tissue has been examined histologically from a few hours after injection to several months, showing gradual development of plastic exudate in the recently injected cases to fibrous tissue in later stages; in its early stages the greater part of the tissue consists of young fibroblasts; in the mature stage the fibroblastic tissue has been converted into dense non-elastic fibrous tissue in no way distinguishable from normal connective tissue.

With these facts it seemed altogether proper to attempt clinically to treat hernia by injecting the inguinal canal. To evaluate the merit of the various solutions offered, in was decided to inject the first 75 patients with Pina Mestre solution. The second 75 patients were injected with proliferol, reenforced by tincture of thuja, phenol and alcohol. The third 75 patients are now being injected with sodium psylliate (sylasol).

The Pina Mestre solution contains the following ingredients expressed in percentages: 25 krameria, 16 catechu, 15 rosa canina, 15 rosa centifolia, 14 vaccinium myrtillus, and 15 monesia in 98% alcohol. Proligerol is a distillate from the tinctures of several botanical herbs, to which is added thymol, tannic acid and benzyl-alcohol. The thuja mixture consists of Lloyds specific tincture of thuja 25%, phenol 50%, and alcohol 25%. This is allowed to stand for 48 hours, and then decanted. Sodium psylliate (sylasol) is a 5% sodium salt from the fatty acids of oil of psyllium.

Before beginning the treatment of patients, rabbits were injected intraperitoneally with 0.5 to 3.0 cc. of Pina Mestre solution. The rabbits showed only slight reactions. When Autopsied several days later, none of them showed any peritoneal reactions or adhesions except the rabbits receiving the 3.0 cc., and those reactions were only mild congestion of the peritoneum and intestines.

It is absolutely essential to know what type of hernia is suitable for injection. A hernia must be completely reducible into the abdominal cavity, and held at all times by a properly fitting truss. Strangulated or incarcerated hernias are of course surgical. Any irreducible mass or suggestion of a mass on the inguinal canal contra-indicates injection. Undescended testicle and hydrocele are also surgical conditions. Asthma, hay fever, severe bronchial conditions, prostatic hypertrophy, urethral strictures and any condition causing straining during urination, should be relieved before attempting treatment by injections or by operation. Overly stout patients with large pendulous abdomens causing increased intra-abdominal pressure, should be reduced before treatment. Hemophilia contra-indicates injection. Diabetes, tuberculosis, hyperthyroidism and syphilis may be treated, depending on the severity of the condition. Age is no contra-indication. One of my most gratifying results was obtained in a man 84 years of age, with a large direct hernia of 14 years standing. During the middle of the treatment he went through an attack of bronchial pneumonia, without damage to the proliferation already obtained. Six weeks later the treatment was continued to a successful conclusion. Femoral, umbilical and incisional hernias may be successfully treated but must be carefully selected. Due to adhesions and no definite sac formation they are often not suitable. Patients with nephritis, cardiac disease, high blood pressure and other condition causing debility, making them "poor surgical risks", can be safely treated.

Any surgeon using the injection treatment her hernia must know the technique of fitting trusses, and the type of truss for each individual case. One type of truss is not suitable for all patients; fitting left to the layman is almost invariably over the external ring, instead of over the internal ring. Not 10% of the

* From the Department of Surgery, Southern Pacific General Hospital, San Francisco, Calif., by FRANK R. GIRARD, M.D., San Francisco, California; read before the Arizona State Medical Association, April 23-25, 1936, Nogales, Ariz.; published in *Southwestern Medicine*, June, 1936.

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millions of trusses worn are properly adjusted. A semi-frame truss with counter pressure over the gluteal region gives as a rule the most efficient pressure and the greatest comfort. Elastic trusses are not to be used. The truss should be worn tight enough to cause an indentation or depression over the inguinal canal to approximate as much as possible the anterior and posterior walls of the canal during the solidification. Occasionally a truss too tightly fitted, especially in double hernia, will cause edema of the scrotum and enlargement of the cord. The truss should be worn day and night next to the skin for at least one week before beginning treatment, allowing the patient to become accustomed to sleeping in it, and the surgeon opportunity to observe if the hernia is held properly reduced, and if the skin tolerates the pressure. The truss is worn during the treatment, and for one month afterward. It is then removed at night, but replaced before the patient gets out of bed and worn during the day, from four to six months, depending on the patient's type of work. If blisters develop under the pad they are easily treated with compound tincture of benzoin or dusted with bismuth formic iodide powder.

Occasionally a large scrotal hernia is impossible to retain properly with any type of truss. These cases I have put to bed, foot of bed elevated, and given them four to six injections daily; then the truss will hold the hernia perfectly reduced, and I finish the treatment in the usual manner.

By whom should this type of treatment be used? Its apparent simplicity is its greatest drawback; the method requires as much, if not more technical skill and surgical judgment than the operative method. It should be employed only by men, surgically trained with knowledge of the anatomy and pathology of the inguinal region, and with accurate diagnostic ability. Unfortunately men seeing only an occasional hernia and who would never dream of doing a herniotomy, will be tempted to employ this treatment, perhaps with danger and disappointment to the patient, and discredit on the method. It is as much a surgical procedure as is operation. The technique is as definite and exacting. The same knowledge and skill is required; the difference is that syringe and a solution are used instead of scalpel and sutures.

With the patient reclining on the treatment table, the pubic hair is clipped. The table is tilted to a moderately steep Trendelenburg position, and the internal ring is located two cm. up from the middle point between the anterior superior spine of the ilium and the spine of the pubes. This area is painted with merthiolate or tincture of iodine. A five cc. luer syringe is used with two needles. The needles should be from 1.5 to three inches in length, depending upon the thickness of the abdominal wall, 22 gauge, and short beveled. The skin is pinched up, and the needle inserted at right angles to the abdominal wall, down to the fascia of the external oblique muscle. Then with steady firm pressure, using the finger touch and not the weight of the arm, the fascia is punctured; a sudden "give" like the puncturing of the head of a drum is felt. The needle is advanced .25 inch further, and the tip of the needle is then in the region of the internal ring. This is demonstrated by free mobility of the point of the needle. Then aspirate to make sure that a blood vessel has not been punctured; inject three cc. of two per cent novocaine. The syringe is detached from the needle which is left in situ. The second needle is then attached to the same syringe and four cc. of the sclerosing solution is drawn into the syringe. After a lapse of five minutes, to give the novocaine time to act, the syringe is attached to the needle in the internal ring; again aspirate to make sure that the needle has not changed position; inject the solution very slowly.

At times patients will notice a warmth in the area injected, and sometimes singing in the penis and scrotum. This is due to irritating of the ilioinguinal nerve; it lasts only a moment and is not severe. If during the injection the patient should complain of severe pain ascending toward the umbilicus, the injection should be immediately stopped because that generally means that some of the solution has entered the peritoneal cavity. If this should happen, abdominal cramps and nausea may ensue, but rarely lasting more than one-half hour. After the needle is withdrawn, the injected area is gently massaged with a small gauze sponge. This frequently increases the hot feeling for a moment. A 0.5 cm. square of adhesive plaster is placed over the pucture as a marker. The patient remains quiet for about 10 minutes. The truss is then carefully adjusted. The table is leveled, and the patient is helped to the upright position by supporting the shoulders. This takes the strain off the lower abdominal muscles. The

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truss is then inspected with the patient standing. He then returns to his work.

The treatments are given three times weekly, but may be given daily if the reaction is slight and time is a factor. Occasionally a patient has to miss a treatment, which is unimportant. Some patients complain of lameness in the inguinal region for 12 hours following a treatment, but it rarely incapacitates for work.

I like to begin the treatments at the internal ring for the following resons: The internal ring is the most important area to obliterate, and the sooner the better. With the internal ring narrowed, the truss can be better depended upon to keep the intestines within the abdominal cavity. If the external ring is injected last, a finger pushed through the external ring into the canal, can feel the needle tip and assure one that the injection is being properly placed. Then the cord lying posterior to the finger can not possibly be injected. This gives better control of the injections. After the first injection at the internal ring, the canal is injected, the injections being given about 1.5 cm. apart, toward the external ring, with the finger in the canal whenever possible. Several injections are given as a routine into Hesselback's triangle, and the last at the external ring. The internal and external pillars and the arch are injected with the finger pushing the cord out of the way.

I have found 12 treatments on the average to be necessary, although some patients may require 15 or even 20, and others only eight to 10. There is a marked difference in the way patients react regarding proliferation; the Negro proliferates rapidly because of the tendency to keloid formation. The average patient receives three injections at the internal ring, four in the canal, two in Hesselback's triange, and three in the region of the external ring. In direct hernias the injections are given first in Hesselback's triangle, with a finger in the external ring as a guide. The edge of the defect in the conjoined tendon can often be readily palpated and elevated a bit, and injected down to the tip of the finger. In treating direct hernias, a few injections are always given into the inguinal canal and the internal ring.

I use the thuja mixture to enhance the proliferating action of the other solutions; that it does this is easily demonstrated. The thuja mixture is given with a tuberculin syringe, and never more than six minims of this powerful mixture for an injection. The thuja is used at every fourth injection. For the patient receiving 12 injections, the 4th, 8th and 12th injection would be the thuja solution. When using sylasol, no local anesthetic is needed. An injection consists of three cc. The same technique is used as with proliferal, but not thuja solution is used with sylasol.

A method of injecting has been advocated whereby the needle is inserted several cm. above the site of injection. The needle is inserted into the skin at about a 45° angle. The point of the needle is directed downward toward Poupart's ligament. The peritoneal cavity is supposed to be less apt to be entered than when the needle is inserted at right angles to the skin. It is supposed to be a safer method for beginners. That may be true; but the cord rests on Poupart's ligament, and is more apt to be damaged by angular insertion of the needle than by injecting into the center of the canal.

When treatment is finished, we expect to have the following changes in the inguinal canal: The external and internal oblique muscles which have been separated from the transversalis fascia by the protruding bowel are now solidly welded together with the cremaster muscle and the spermatic fascia, and the loose areolar tissue in the inguinal canal. A firm dense fibrous inguinal splint is developed. Except that the hernia sac has not been removed this should give the same end results as an operation would have given. In many patients the sacs are obliterated I believe through chemical inflammation extending from the surrounding tissue fusing the walls of the sacs. Eventually the sacs are transformed into fibrous bands.

At autopsy have been found not infrequently hernial sacs extending onto the cords. The men with these were never conscious of having had hernias, and never had symptoms thereof because the necks of the sacs were so constricted that the abdominal contents were never able to enter the sacs. By narrowing and solidfying the inguinal canal we do exactly the same to the sacs. Unless the necks of the sac can be completely obliterated, treatment by injection will fail.

I recite in detail an example of failure: A patient had a large left labial hernia. She was given 12 treatments, and a firm proliferation in the inguinal canal was produced. Two months later she returned with a definite bulging and an impulse on coughing, but smaller than previously. She was given 12 more treatments. By this time the induration felt almost cartilageous. After several months, wearing her truss all the time, she still had a small hernia. She was opposed to an operation, and for the third time we attempted to close the canal by injections. She received 36 injections without closure of the hernia sac. She finally submitted to operation; the fascia of the external oblique was densely adherent to the underlying muscles; a large markedly thickened sac was dissected free; surrounding three-quarters of the circumference of the neck of the sac was a firm mass of fat about the thickness of the end of one's thumb which prevented the proper closure of the internal ring and obliteration of the sac. The density of the muscles and Poupart's ligament was astounding, it being with considerable difficulty that the needle could be introduced into them.

Microscopic sections. Dr. A. M. Moody, pathologist, reported upon microscopic sections as follows:

The scar tissue which has developed in sections of injected hernia is very dense, relatively thick and markedly hyalinized. This condition has occurred with a minimum of cellular reaction.

With operation only, a recurrence means a second operation — always more dangerous and more unsatisfactory than the original one.

The number of treatments is determined by the size of the hernia defect, the response of the tissues to the injected solution, and the amount of reabsorption which takes place after the first course of treatment has been given. I believe that the treatment should be considered in two stages. The first stage is the original course. The second stage is the period of observation, lasting from one to one and a half years. Patients should be carefully examined every two months after the truss has been removed. In an area is found which shows evidence of weakening, a few more injections should be given. Not until the patient has been under observation for from one year to 18 months can one feel sure that a permanent cure has been obtained.

In our series of 174 cases 19, or approximately 11%, were recurrences following herniotomy. I believe that with the combined technique of operation and injections, if recurrence is threatened, that the necessity for operating on a patient the second time for hernia will be eliminated.

Many complications have been mentioned as likely to result from the injection treatment none of which, in our series of 174 hernias treated and under treatment at present, have we encountered. Infection, abcess formation, peritonitis, atrophy of the testicle and sterility have all been noted as possibilities. The one common complication has been moderate swelling of the cord, lasting from a few days to several weeks, but always subsiding, leaving no permanent damage. A few cases have complained of swelling of a testicle for seldom more than 24 hours. One must be particularly careful when injecting the internal ring because of the proximity of the anterior crural nerve and the femoral vessels. When injecting this area I tell the patient to notify me immediately if there is pain running down the anterior surface of the thigh toward the knee. This happened in two early cases with no permanently bad result.

The injection treatment of hernia was first begun at the Southern Pacific General Hospital in July, 1934. At the time this paper was written a total of 174 cases had been treated or were under treatment. There were 129 indirect hernias, 19 direct, four scrotal, 19 recurrent following herniotomy; two femoral and one umbilical. In this series we had one definite failure, requiring operation. Eleven have required further treatment because of weakening of the canal due to complications such as vomiting, loss of weight from peptic ulcer, severe spasmotic coughing from tuberculosis and asthma, influenza, and improper wearing of a truss.

I know of no type of treatment in which cooperation of the patient with the surgeon is more imperative, especially regarding the wearing of the truss properly for the required length of time. The majority of our patients are rather ignorant laborers, and it is difficult to make them understand the importance of the

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properly placed and well fitted truss on the final result.

Considerable judgment must be used in accepting patients suffering from peptic ulcer, arrested tuberculosis, asthma, etc. They often appear to be in good physical condition and well suited for treatment. After the treatment is finished they may develop severe symptoms from their complicating disease which will have an unfavorable reaction on the hernia treated. I speak feelingly on this subject because I have recently examined two patients who apparently had satisfactory closures of their inguinal canals but one man developed an ulcer attack, with severe vomiting, hemorrhage, and loss of nearly 40 pounds in weight. During the attack he removed his truss and when he appeared at the clinic some months later, his truss was absolutely useless, being now entirely too large, giving no support to the inguinal canal. Consequently there was a slight return of his direct hernia. The other patient returned 14 months after the finishing of his treatment with slight bulging at the external ring of one week's duration. He stated that for years he had been receiving 50 per cent disability compensation from the Government for arrested tuberculosis associated with asthma, and that he had frequent violent coughing attacks, so severe as to cause emesis.

I cite these cases to show that while patients appear to be in good physical condition, histories should be carefully taken to determine if any condtion might contro-indicate the injecttion treatment.

Recently all patients who had received treatment longer than six months ago were requested to report for examination, or to have their company doctors examine them if they lived some distance from San Francisco. To date, those who have responded show satisfactory closure of the inguinal canals, but I expect to find some who require a few more reenforcing injections. It is most gratifying to note the interest in this method of treating hernia by the more conservative surgeons, medical schools and clinics, who several years ago showed little interest in it.

Those of us who are pioneering this work should give every aid to those wishing to test this treatment seriously, and in a thorough scientific manner. Every surgeon using this method on a large number of patients likely will have something constructive to offer from his observations. With the right men and institutions using it, a great deal of the prejudices will be removed. Much experimental work must yet be done in the matter of solutions, number of treatments required, and in judging the type of hernia suitable for this treatment. Should it be confined, as some men think, to small indirect, incomplete hernias, or should treatment of all types of reducible hernia be attempted? Refinements in technique and the types of trusses to be used must also come in for serious consideration.

We look forward in the not too distant future, when the surgeon will find this method of treating hernia, a most valuable aid to the operative treatment, especially when used after operation to strengthen a post-operative weakening of the canal, thereby practically eliminating the need for re-operation.

CONCLUSION: 1. A rational method of curing certain types of hernias is offered by the injection method.

2. It should be used only by men, surgically trained, with knowledge of the anatomy and pathology of the inguinal region and with accurate diagnostic ability.

3. The method is safe when used by experienced operators, but there are potential dangers when used by men with little knowledge, of the exacting technique and, of hernia in general.

4. The numerous complications mentioned are mostly theoretical, and have not occurred in my series of 174 hernias.

5. Many patients, poor subjects for operation, may be treated by this method and rehabilitated to useful occupations.

6. The value of the operative treatment of hernia can be greatly enhanced by the use of injections when a post-operative weakening is threatened or recurrence develops.

7. Economically there is a great saving of time and expense because loss of time from work and hospitalization are not required.

8. The wearing of a properly fitted truss for perhaps six months after treatment is most essential.

9. A careful check-up every two months for 18 months following the completion of the treatment should be made, and a few re-enforcing treatments given when signs of weakening are discovered.

10. Investigation of this method by hospitals and clinics where large numbers of hernias are treated will give this treatment the dignity and standing that operative procedure has.

Discussion

DR. BROWN: Both speakers are to be congratulated; they have dived into a here-to-fore nearly forbidden field and done it in a conservative, scientific convincing manner. I have no criticism and relatively little to add to what they have said.

My interest in the injection treatment of hernia is through a sequence of events covering many years. My father had hernia. I recall as a small boy the trouble he had with his trusses. He would never consider an operation. In the course of years many persons similarly afflicted with hernia and trusses who would not be operated have been seen. Many persons who had operations which are failures are known.

Some 10 years ago I developed a hernia which was operated by two of my very good friends in whom I have the utmost confidence and who I am sure did the best job possible; through the weakness of my own tissues it recurred in a short time. A year later the same hernia was repaired by a competent St. Louis surgical friend. It has held although he

doubted that it would; but within the year I have had several attacks of pain indicating weakness in the lower portion. About one and a half years ago a weakness appeared in the right internal ring. About this time I heard of the Pina-Mestre solution which was being distributed in this country by a son of an old school friend of mine; I was hoping to get in touch with him when one day Dr. Ralph Palmer told me of his interest in the injection treatment of hernia and that he was gathering data upon it. He soon gave me six or seven injections and apparently I am completely cured. No time was lost; I had no argument with my insurance companies. The two operations had caused me to lose about three months' time and to have considerable discussion with my insurance carriers.

I studied with Dr. Quillan of Chicago last fall and recently wrote him for a few facts. He has had about 10 per cent recurrence; they have been easily controlled by additional treatment. He would probably agree with Dr. Girard that they are not actual recurrences but merely undertreated cases. Dr. Quillan has used several solutions, some of which he made, but I believe in the main he uses proliferol or sylasol. Dr. Quillan says that it is not necessary for the needle to enter the hernia sack. In one or two cases to be operated he injected the sclerosing agent mixed with a dye upon the hernia sack and found the colored solution within the hernia sack. Dr. Quillan has treated 344 cases. He has had no complications. He has seen two cases with sloughs, due to the sclerosing agent's being deposited subcutaneously instead of beneath the deep fascia. Sometimes patients get soreness in a testicle or swelling in a cord, but these are not serious. I had slight soreness in the testicle for several months after treatment.

Any material to be injected into a person first should be tested to see if that person is sensitive to it; also the induration in the skin from the test will give the physician an idea of how that patient's tissue will proliferate. I had reactions in my course of treatment on two afternoons; I was achy and tired and I had some pain. No reaction was incapacitating.

I have treated several cases, I believe successfully, with no loss of time and discomfort to them.

I belive Dr. Palmer's idea to take care of hernia occurring in industry is an advanced idea and one well worth considering. It seems to me that it will save money for the industrial commission and will be of great help to the men needing the treatments. I feel it is a distinct honor to have been asked to discuss Dr. Girard's and Dr. Palmer's papers.

DR. REESE (California): In our state we have a compensation law similar to that in Arizona. Hernia offers three factors for consideration; The patient, the corporation paying the bills, and the physician, each interested in the subject. Experience has proced that insurance carriers are magnanimous in considering hernia. Hernia is comparable to a tumor in a woman's breast; congenital weakness and outside contributing factors lead to its development. In hernia, chronic constipation and urinary disturbances are contributing factors (and severe coughing.—Ed.). One thing which insurance companies definitely pay attention to following a supposed traumatic hernia is nausea, vomiting, sweating, and slight shock. Such symptoms with the occurence of the hernia support its traumatic origin. Dr. Palmer's suggestion and plan for the care and treatment of hernia in Arizona is most revolutionary and offers the patients all the breaks. If Arizona is to assume full responsibility for the treatment of these cases she must also prepare to assume full responsibility for any and all complications that may arise. Time is a great leveler. The injection treatment for hemorrhoid is not all that is promised to be; many are now avoiding this treatment. The treatment is far from fool-proof. Sterility has been recorded in several cases as a result of the treatment. The choice of solution must be a careful one if sloughing is to be avoided.

DR. FERGUSON: I have one question. Nothing has been said as to the type of work the patient may do while undergoing treatment.

DR. GIRARD: I appreciate the discussion. In reply to Dr. Ferguson: If a patient is doing heavy work, such as lifting, we request that he be given lighter work during the treatment. It is purely a matter of judgment. Dr. Palmer's plan is most revolutionary and altruistic. I think, however, that industry may be shown that there is a responsibility for them to shoulder. If hernia occurs during a man's work, industry certainly should care for it. This care is most expensive in the majority of states. In New York, for example, the records of the department of labor show that in the period from 1926 to 1930 the total cost for temporary disability was $76 per person; between 1930 and 1934 the cost rose to $217 per person, with an average medical cost of $170 per person. New York compensates 40% of a man's wages leaving a 60% uncompensated loss. Adding the medical cost, plus 40% compensation, plus the loss to the man himself, New York has a total cost average of $687. The figures for California are not available. In San Francisco, however, the average treatment time is 19½ days which at $4.00 per day represents an approximate cost of $80. The operating cost is $5.00. Next we add a 46-day period of total disability at $5.00 per day making a loss of $230. Thus we can see that the average cost per person for this treatment is $315 for San Francisco. Only time can tell how permanent the results from this treatment may be. Referring to the 10% recurrence as Dr. Brown quoted from Dr. Quillan I would say that this represents under-treatment rather than recurrence. Some respond beautifully to a few treatments and the physician feels enough treatment has been given, while others need many treatments to close a hernia. I agree that thuja is dangerous but can give good results if used carefully.

Of our 175 cases we have never had a slough nor an infection. Men must exercise skill and care with the injection just as in surgery. One great advantage of this treatment is that potential hernia may be easily and successfully treated with such a degree of success that a man may secure employment otherwise denied him.

The injection treatment of hernia is comparable to the injection of varicose veins. At first, in the treatment of the veins, there was a multitude of solutions, now but one or two remain for general use. The same will be the case with solutions now being advocated for hernia. We are being constantly bombarded with various solutions for this purpose. These have not been sufficiently tested; we have only the word of the manufacturers that these solutions have the virtues claimed for them. Proliferol seems effective and worthy, whether it will become of permanent use only time can tell. Recurrences may be due to improperly placed trusses. I have in mind a case where the truss was found to be over the rectus muscle, fully 1.5 inches away from the inguinal canal. The proper position and placement of the truss is most important. As a matter of sterility, referred to by Dr. Reese, it seems odd that such a condition could occur as tests with animals show no such obstruction. Then, too, should one side become occluded, there still remains the other to function. The statements about sterility have come from men who have not done this work. Men who have done the work report just the opposite. In Minnesota, for example, of 1,500 recorded cases there is not one to demonstrate a sterility, a peritonitis, or an abscess. If this work is not done by men who are surgically minded and surgically trained, trouble will result. Dr. Brown spoke of a tovic condition following his treatments. While such conditions have been occasionally reported, none has been incapacitating, and it is rare that a man is kept from his work.

DR. PALMER: I appreciate the responsibility in the plan we have outlined for the care and treatment of hernia in Arizona. I shall see personally that no industrial case will be referred to a surgeon who has not given much study to the subject and who does not have the time to devote to the work; only the efficient man will be considered. Habits of living might have something to do with the cases of sterility referred to: it seems to be characteristic of men in the high income bracket to experience a period of sterility around the ages of 40 to 50, attributed by many to nothing more or less than habits of living.

It is true that California and Arizona have similar compensation laws. When California made a restricted ruling on hernia, Yuma, Arizona, was literally swamped with hernia cases coming over from industrial California. In turn when our commission made restricted rulings, these patients scurried back to California for treatment. From experience with the injection treatment of hemorrhoids and varicose veins, men who aspire to do this work in hernia must give it careful study and attention and keep the solution where it belongs.

Books

Vocational Rehabilitation And Workmen's Compensation

THERE is little or no therapeutic benefit in cash settlements paid to injured workmen who have traumatic neuroses, according to an investigation made recently by Carl Norcross, Ph. D., of the Rehabilitation Division of the New York State Department of Education. Results of the investigation have been published under the title "Vocational Rehabilitation and Workmen's Compensation," and the report is a follow-up study of 322 workmen's compensation cases throughout New York which were closed by a lump-sum settlement of $1000 or more.

It has been generally accepted in both medical and workmen's compensation circles throughout the country, says the report, that a cash award would help to cure a neurosis. "A careful investigation made a year or more after the settlements has convinced us that the value of a cash award is vastly overrated," writes the author. "It is the settlement of the case, the actual ending of the litigation, which is of value. Whether the final compensation award is paid in one lump or extended through a number of installments makes little difference to the claimant's condition."

The investigation disclosed that 16% of the men had lost a large share of their compensation through unwise expenditures. The men who had no losses were found to have dissipated their funds much more rapidly than they would have under an installment system. Both because there was found to be a wastage of compensation funds, and because there appeared to be no therapy in the settlements, the author has recommended that lump-sum settlements be discontinued.

Dr. Norcross makes a number of recommendations for improving the handling of neurotic cases in the workmen's compensation rooms. He urges that cases be given a more prompt and careful handling. The average neurotic case is open in the workmen's compensation division for nearly three and one-half years, it is said, and much of the delay is unnecessary. The author states that neurotic conditions grow as cases are delayed. He also points out the dangerous policy of permitting claimants to read their own medical reports, or to be present when physicians are testifying, especially in contested cases where there is a difference of opinion.

In New York a compensation case theoretically may always be reopened. The report suggests that it is a poor policy to let neurotic claimants know that when their money is spent that they may try to reopen their cases. The author believes that one of the evils of the existing New York system in non-schedule cases is that claimants must be willing to accept a lump-sum settlement. After a fair offer is made, the neurotic claimant may procrastinate indefinitely by refusing such settlement. Thus the case is delayed, and the patient's mental condition may become worse. The remedy suggested by the report is that the referee, acting on competent medical advice, fix a fair settlement and close the case, with the award being paid in biweekly installments.

To overcome any prejudice the claimant may have toward the insurance company, it is suggested that the money be paid to a State-administered trust fund, which already exists in New York. The carrier could close the case on its books, and the claimant could be told his case is definitely closed but that he would get all his money, regardless of his state of health. The patient would not have to remain sick to get his award.

Provision is made for permitting the claimant to get an advance on his compensation for any necessary purpose, including rehabilitation himself on a farm or in a small business.

Copies of the report may be secured through the publisher, The Rehabilitation Clinic, 28 East 21st Street, New York City. Price $1.00.

Industrial Environment And Health

INTERNATIONAL Labour Office, Geneva, has just issued a 336-page booklet entitled, "Industrial Environment and Health". This, pending a more comprehensive review, may be described as a sort of factory inspector's manual telling how certain tests can be made in the course of industrial inspection to determine such factory conditions as light, humidity, temperature (maximum and minimum), ventilation, dust accumulation, noise, and other conditions of health and hygiene of the given establishment.

Silicosis Legislation in New York

THREE acts looking forward to the elimination or control of silicosis as an industrial hazard, and providing for limited conpensation for workers disabled by this disease were passed by the New York legislature.

The principal provisions of these acts are briefly as follows:

Provision for special and limited workmen's compensation to employees disabled by silicosis or other dust diseases.

Prohibition in so far as possible of compulsory preemployment examinations of wage earners.

An allotment of $50,000 each year for five years to be spent for research studies on dust and other occupational diesases from the viewpoint of prevention, including the design of equipment, its poeration, and studies of efficiency of apparatus in use.

Making mandatory the use of approved dust-eliminating devices and methods on all public works, includin ghighways and construction, both indoors and outdoors.

Directing the Industrial Commissioioner and the Industrial Board to promulgate industrial code rules and regulations for the effective control of silicosis and similar diseases in all industries and operations wherein silica dust and other harmful dust hazards are present.

Appropriation of $100,000 for administrative expenses and for employment of examining physicians and research consultants.

Diagnosis and Treatment of Traumatic Injuries of Intra-Abdominal Viscera

THERE is probably no diagnosis requiring more finesse or surgical acumen than the rapid differentiation in the diagnosis of the traumatized abdomen.* Rapidity is important because all mortality statistics indicate that the longer surgical intervention is postponed, the more acutely rises the mortality rate. Yet the accurate decision as to whether the surgical abdomen is present can only be made after certain definite diagnostic steps have been taken. Both clinical and laboratory methods occupy a very definite place in offering aid to the surgeons in the determination of intra-abdominal pathologies; however, in our estimation, accurate observation, coupled with a keen clinical sense and truthful scientific interpretation, is definitely more important than any laboratory aid. All diagnostic methods used in their utmost still leave a very definite number of cases that will only be completely diagnosed following abdominal section or at the autopsy table.

That definite progress is being made in the treatment of such surgica condition is very much evident by a study of the mortality statistics over a period of years. We find that prior to 1890 the mortality of these cases was about 90%; by 1900 the mortality had fallen below 75%; and early in 1920 many authors report a mortality of around 50%. In the past few years Dean Lewis and Trimble, in reporting 140 cases of "subcutaneous abdominal injuries," show a mortality of 30%[2].

The usual classification of traumatic lesions of the abdominal viscera are open or penetrating wounds, and subcutaneous injuries—according to the mechanism of the traumatic force. The former is usually caused by stab or gunshot wounds and the latter is the result of blows to the abdominal wall or indirect violence. The difference in the treatment of these two types of injuries is quite marked, as is also the ease with which the accurate diagnosis is made. In the penetrating wound the severity of the injury is usually quite apparent, as is also the general direction of the underlying pathology. In subcutaneous injuries this is not the case—the injury varying as to the force and direction of the trauma. Inasmuch as this paper will deal primarily with subcutaneous injuries resulting from blows to the abdominal wall, suffice it to say in regard to penetrating wounds, that in the great majority of cases it is well to adopt the general policy of abdominal section with complete intra-abdominal exploration at the earliest possible moment.

Time-consuming methods of making a differential diagnosis of pathologies resulting from blows or direct violence to the abdomen should not be tolerated. Someone has reported sixty-one possible injuries to the intra-abdominal viscera. To attempt to differentiate the exact pathologies which may be found on ab-

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*JOHN BARON FARLEY, M.D., Pueblo, Colorado, read before the Spring Clinics of the Pueblo County Medical Society, April 29, 1936; published in *Colorado Medicine*, August, 1936.

dominal section, while assuming the role of ultra-scientific surgery, in the last analysis may prove only pseudo-scientific insofar as the life of the patient is concerned.

It would hardly seem necessary to mention that the most important initial step toward diagnosis is to obtain as accurate a history as circumstances will permit, particular attention being given to the amount of force and the direction in which the force was applied.

The differential diagnosis between shock and hemorrhage is undoubtedly the most important and probably the most difficult. The blood count which is of great value here should be repeated at hourly intervals. While a high differential count helps very little in differentiating a peritonitis from an intraperitoneal or retroperitoneal hemorrhage in cases that have not been hospitalized soon after the injury, it is of extreme value and definitely does show that there is more than hystria in a restless patient who is seen early and who has been thought to have been trivially injured. Primary shock is often mild following abdominal injuries; secondary shock is as severe and terrifying as the primary shock is mild. The differential count is of great value here because the blood changes appear quite early, sometimes several hours before the secondary shock.

It will perhaps be best to consider the different pathological entities in the order of frequency with which they occur: (1) Without doubt, mesenteric tears are most frequent in occurrence. (2) Next in order is retroperitoneal hemorrhage. Seldom does the surgeon open the traumatized abdomen without finding one of these two condition, in addition to whatever other injuries it may contain. (3) Next in order comes liver tears, with or without damage to its ducts. (4) Injuries to the spleen and urinary bladder stand next in order to be closely followed by (5) injuries to the gastro-intestinal tract, the most common site of pathology being the lower jejunum and upper ileum (6) Next in frequentcy comes the duodenum and stomach. (7) Last are the pancreas and colon. It is said that perforation of the colon due to non-penetrating trauma is so extremely rare that it is not listed in the "Index Medicus."[2]

Mesenteric injuries consist of tears and hematoma formation. The tears resulting in hemorrhage may require a simple suture or even resection of the bowel.

Retroperitoneal hemorrhage often requires no surgical treatment. As the presure becomes increased the hemorrhage is arrested. However, there always remains the possibility of later abscess formation. The functions of the abdominal and lumbar sympathetic systems, lying retroperitoneally, are often embarrassed to the extent of giving a false typical picture of a ruptured viscus.

Injury to the liver, which is the most frequent of the large organs to to injured, generally results only in hemorrhage. This hemorrhage may be large or small, although at times when there is injury to the ducts there may be and generally is a resulting chemical peritonitis from the extrusion of bile. The size of the tear may vary from a small laceration a centimeter in size to the complete halving of the organ. In general the treatment consists of the close application of a vaseline tamponade to the bleeding areas. Suturing the organ is quite unsatisfactory because of mechanical difficulties. The old bugaboo of liver abscess resulting from tamponade was dispelled by Prey and Foster who found in going over fifteen years of autopsy records at Denver General that there was not a single case of liver abcess resulting from this treatment.[3]

The kidneys, which come next in the order of frequency, possibly hold their high place due to the ease with which the diagnosis is made. Squeezing of the kidney, or even a small tear, will often result in blood in the urine. A tear which penetrates into the calyx will of course produce a severe hematuria. Unlike severe injuries to those organs contained within the peritoneal cavity, in the case of kidney injury the conservative watchful waiting is strictly in order. The reparative and regenerative power of the kidney is almost unlimited. Hemorrhage from a ruptured kidney is rarely severe enough to require treatment on short notice unless the pedicle has been torn. Pressure that is produced within the perinephritic space will generally be sufficient to control the leakage. A frequent end result of large hemorrhages of the kidney is a perinephritic abcess. Lithiasis such as might prove dangerous to life should be regarded as a significant possibility.[4] Acquaintance with this fact is of the utmost importance forensically in the adjudication of personal injury suits.

More apparent than real is the protection afforded the spleen by the lower left ribs. Slight trauma will at times cause great damage to this organ. The spleen is definitely less elastic than either the liver or the kidneys, and the latter two are possessed of a tough limiting membrane, or capsule, which tends to make these organs more elastic. The limiting membrane of the normal spleen which is thin and friable, does not deserve the name of "capsule." Hemorrhage from the spleen may be terrifying, and in such cases the only efficient treatment is splenectomy. The vaseline tamponade may be used in small lacerations, but the danger of secondary hemorrhage should be borne in mind.

Urinary bladder injuries may generally be diagnosed with only the aid of the catheter, and they may certainly be confirmed by the use of the cystoscope. Sever pain over the region of the bladder coupled with a bloody urine or the inability to withdraw urine is almost pathognomonic of tear. While these tears usually allow the urine to enter the peritoneal sac, it should not be forgotton that they may also lead into the retroperitoneal space. Immediate surgical repair is indicated in either condition, and in the exact localization lies the value of the cystoscope.

Lower jejunium and upper ileum injuries, as well as any other gastro-intestinal perforation, are possibly best diagnosed with the aid of the x-ray which shows free air in the peritoneum. Clinically this is manifested by hepatic resonance. Treatment varies according to the size of the perforation which may require a simple suturing or an extensive resection.

Duodenum and stomach injuries as well as bladder injuries were shown by Reed in 1933 in a very conclusive manner to occur only when these organs are distended.[5] Therefore, the history of eating or drinking just prior to injury is of the utmost importance. The bursting of the full stomach or duodenum produces a squirting of the contents throughout the peritoneal cavity, which causes at the moment of injury a generalized peritonitis of both septic and chemical nature. Such a patient is in immediate severe shock with a rigid, tender upper abdomen which is enormously distended. Massive hemorrhage from the liver or spleen will often give much the same picture as a fulminating chemical peritonitis, but because laporatomy is indicated for either hemorrhage or peritonitis, the need of delay for extensive blood study seems unnecessary and time-consuming.

That the pancreas is an extremely well-protected organ and that it is seldom injured is shown in a report of 9,500 autopsies at the Pathological Institute of Leipsig over a period of seven and one-half years in which only one grossly visible rupture of the pancreas was found. Stern, after a most diligent search, did find 100 reported cases; of these, only twenty-eight existed without associated injuries of other viscera.[6] It might almost be said that when there is an injury sufficient to produce a rupture of the pancreas the other visceral injuries will be so severe as to make recovery almost a miracle. However, should a patient with a pancreatic rupture recover he may be cheered by the fact that Umber reports that there is no relation between true diabetes mellitus and pancreatic trauma.[7]

When to operate is an extremely interesting question—and the most difficult to answer. Watchful waiting assumes the proportion of a diabolic temptation in the light of a survey on its disastrous effect on the mortality rate. When we consider that it may be dogmatically preached that all cases of a perforating lesion of the gastro-intestinal tract, all massive hemorrhages of the liver or spleen, and all tears of the urinary bladder must be operated to live, it seems but a natural conclusion that once the primary shock is under control, the sooner the better.

If one or more hours of efficient shock treatment with its stimulants and fluids have passed and the patient remains restless and anxious, with a rapid and feeble pulse and a subnormal temperature; if the extremities and tongue remain cold; with the abdomen that is rapidly becoming rigid and is tender to the slightest palpation; and if there is reasonance in the hepatic region, and possibly iliac or flank dullness; or if neither urine nor flatus has been passed—with these findings or with a majority of them, providing the systolic pressure has begun its climb following the primary shock and is now hovering around 85 or above, the verdict then would be that the operation should be performed without waiting for other symptoms.

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Mental and Emotional Rehabilitation of the Injured Worker

TO DISCUSS the mental attitude of the worker is a monumental task, one to which a lifetime could be devoted.* Thick and dusty volumes covering the subject fill whole libraries; nevertheless, in all humility, I shall try in the next few minutes to give you a comprehensive estimate of the many factors that help to determine the mental attitude of the working man toward his job, toward any injury he may receive, toward his employers and toward the company carrying the compensation insurance required on his particular job.

It must be remembered that there are as many mental, or shall we say emotional attitudes as there are workmen. Workers protected by compensation insurance are human beings and therefore as varied in type as the pranks of nature It is characteristic of human beings to be thus varied and one need not be a physician or skilled specialist to recognize it. A workman can be damaged emotionally just as readily as physically, perhaps more so, and it is as important to rehabilitate him emotionally as physically.

In the past half century medicine and surgery have made great strides in eliminating the death-dealing infections and epidemics of a few generations ago. The merciful skill of the surgeon's hands has restored much usefulness to a workman's mangled limb or saved the vision of a threatened eye. Very decidedly, there has been rehabilitation here. Then, a growing, pushing civilization has added a new element of rehabilitation for the workman. He is the safety engineer. His foresight and skill have provided guards about machinery, adequate lighting, respirators to protect against dust and noxious fumes. He has placed rubber treads on the stairs, ventilators in the factories and offices, and water coolers in the corners.

At a casual glance it would seem that everything possible has been done to rehabilitate the worker. It would seem that he is in a working man's paradise, when judged by the standards of fifty years ago. But there remains a vast amount of rehabilitation to be done, and it seems to me as a psychiatrist that this job is chiefly one of emotional restoration.

To be logical, we must review the position of the working man even before he is hurt. Almost without exception, our workers reside in cities. The greater part of his time is spent in some large factory or foundry and usually here he leads a sedentary existence. His work is often dull and monotonous, even if skilled, and he often has to renounce the age-long habit of using his muscles to gain a living. If he is employed in the so-called white collar jobs, he is no better off. The economic basis of our civilization is such that all these workers are entangled in the great web of dependency. This at once establishes a mental attitude in the working man that may prove a difficult factor to surmount if rehabilitation is necessary following any injury or illness.

If injury or sickness do occur, despite the preventive efforts of your safety engineers, then at once rehabilitation must commence, and remember that this reconstruction must be phychological just as much as physiological. In fact, large filing cabinets in my office, filled with such histories, have brought me to the conclusion that emotional rehabilitation is perhaps the most important element in the restoration of the patient. As I have just said, the dependency of the worker on his job has created a mental attitude toward his work, his employer and the physician who may take care of him, even before an accident occurs. A natural suspicion and antipathy, nourished by fear, has already become rooted in the ground work that is the workers' mental attitude.

Fear and worry have killed more people than organic disease ever shall. Immediately after the injury fear asserts itself and at once colors the reaction of the worker. Besides the immediate pain and physical discomfort that heckle the patient, there is also the fear of permanent or partial disability eventually, and the immediate worry over economic loss. It is very human to wonder "what will become of me and my family?" Right at this point mental rehabilitation must be instituted. The attending physician must assure the injured worker that his hurt is not too serious and that in due time he will be back at work, no worse for his unfortunate experience. Even if the doctor suspects that damage is quite extensive and that there will be permanent disability, this is not the time to tell the patient. Worry about his physical condition will greatly retard the injured workers' progress. The patient subconsciously will decide, "Why should I get well at all and be a cripple and a burden to myself and to my family?" Maybe he is right—who of us can say?

All men are susceptible to suggestion and unfortunately to the type of suggestion that is harmful to them.

False sympathy may drive an injured patient into a true neurosis where he becomes depressed and introspective. He comes to live in a world of fantasy where he is imbued with the belief that he is unable to work or play. I have seen hundreds of emotional wrecks following injuries in which their difficulties could be entirely ascribed to some unfortunate remark of the attending physician, the nurse or a relative. "The Doctor told me I had a skull fracture. I know what that means. I knew John Smith who went insane from a skull fracture." The nurse may have said to the patient, "It will be a long time before you can work again. I have had patients who have never gotten over trouble like this." The poor wife may remark in the patient's hearing. "John is so sick. I am worried about him. I am afraid he will never get well." When the neuropsychiatrist first sees the patient, the damage of pessimistic suggestion has usually already been done and often, in spite of all the explanation and helpful advice, the patient will persist in holding to these beliefs which are harmful to both him and his family. He unconsciously resists the help that would aid him in finding his normal goal—his happiness.

More must be done to reassure the injured, however, than the soothing words of the attending physician. The employer, or his representative, must appear at the hospital as soon as feasible and show a brotherly, sympathetic and reassuring attitude. Regularly delivered compensation checks, the promise of his old job when he has recovered, well wishes and an attitude of fellow feeling will do much to quiet the jangled nerves of the worried patient. If it is evident that the worker will not be physically capable of returning to his old job, the reassurance of another, maybe lighter work with a living wage, will lift much of the great burden of worry from the shoulders of the injured man. Later, when the patient does return to work, and if the attending physician has prescribed a change of work or even lighter work, this promise must be kept by the employer or the man representing him. This reassuring promise on the part of the employer is a moral obligation and extremely essential to good mental rehabilitation.

It is also good business, even at first glance.

Another step in rehabilitation that has a large phychic influence must be taken as soon as the patient's condition permits. It is complete removal from the influence of hospitalization. Of course, this must never be done until the attending physician feels it is safe. The average layman still feels that if he is confined to a hospital he must be very sick. Organized medicine, through its educational committees, is doing all it can to teach people that hospitals are institutions established for quicker cure and better care of the sick and injured, but such education takes time and we must be patient until it is achieved. Once removed from the hospital and returned to his home, the sick one feels that he is on the mend. He worries less about his physical troubles and begins to feel that maybe after all he can return to society as a useful individual—even able to carry on the work that means livelihood for himself and his family.

* DR. ANDREW I. ROSENBERGER, Milwaukee, addressing Wisconsin Council of Safety, Annual Session, Madison, June, 1936.

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Silicosis and Workmen's Compensation

THERE have been some interesting reactions from the inclusion of silicosis in workmen's compensation acts. After the all-inclusive occupational-disease amendment to the New York Workmen's Compensation Law was passed insurance companies notified the policyholders that their men must be examined for silicosis before the employers could obtain workmen's compensation insurance. When the president of the State federation of labor brought this matter to the attention of the Industrial Commissioner of New York State, the commissioner stated 'that he considered this an extremely undesirable procedure for the following reasons:

In the first place, from the standpoint of the cost of insurance, it makes the workers claim-conscious, and, more important still, it has a serious effect upon the morale of the men by exciting them about their physical condition. Then, too, if men are ruthlessly discharged, they, of course, have recourse to civil action against their employers because of the physical impairment which arose in the course of their past employment.

As you know, these civil actions in the past have resulted in large awards to the workers or their widows or other survivors and have been a large drain upon the reserves of industry. This was one of the factors in the support given by industry to the silicosis bill enacted at the last session of the legislation but vetoed by the Governor because of the high rates which insurance companies said would have to be charged if the bill became law. Although it is true that workers who are members of labor unions have usually refrained from filing civil suits because to do so might have forced their employers out of business and also because the workers prefer wages to uncertain and delayed court awards, if a policy of physical examination and elimination of affected workers from employment is adopted by employers, either voluntarily or under the compulsion of their insurance carriers, it may be that those workers will follow the example of employees in open-shop plants in bringing civil suits. Such a development would be unfortunate in its effect upon industry, employment, and the morale and actual health of the workers. Physical examination of employees who have spent many years of their working lives in an industry would be justified if there were some method of compensation for those wage-earners who, while not disabled in the sense that they are unable to perform work, are found to be suffering from dust disease in some stage. . . .

As to the alarm—real or assumed—with which some insurance companies are viewing the cost of coverage on risks where there may be a silicosis hazard after September 1, and the alarm they are spreading to their policy holders and through them to their workers, may I say that I consider this excitement greatly exaggerated. No workman on September 1 who was able to do a day's work on August 31 would quit work with the idea of bringing a claim before this department and receiving an award. Compensation awards can only be made when an employee is unable to work or else when he can prove he has a decreased earning capacity.

In all probability there will be a silicosis bill introduced next January in the legislature and, if acceptable to workers and employers, will probably be passed. If this materializes, silicosis will only remain within the all-inclusive occupational-disease law from September 1 until early next year, and I cannot conceive how in that time there can be any greater number of claims for silicosis upon which awards would be made by this department and affirmed by the industrial board.

In the meantime it is for all of us who are interested in the prevention of dust diseases for the benefit of both industry and workers to do what we can to make prevention effective.

The results of following the procedure deemed "extremely undesirable" by the New York Commissioner of Labor are illustrated by a case occurring in Wisconsin in 1935; according to the Journal of the American Medical Association* a foundry company had to reinsure its employees. The prospective insurer demanded as a condition precedent to the execution of a policy that all employees be examined by a physician selected by it. Before the examination was made all employees were discharged. Only those were reemployed who were believed to be physically fit for the duties to be imposed on them. Five employees who were not reemployed, because they were not physically fit, were awarded compensation by the industrial commission, under the Wisconsin Workmen's Compensation Act. Thereupon their employer and its insurance carrier brought actions against the commission and the several claimants to set aside the awards. From a judgement modifying and affirming the award in each case the industrial commission, the several claimants, the employer, and the employer's insurance carrier appealed to the Supreme Court of Wisconsin. The Journal gives the decision of the court as follows:

*From "Review of Literature on Effects of Breathing Dusts with Special Reference to Silicosis," part III-B, by D. Harrington and Sara J. Davenport; Information Circular 6892, U. S. Department of the Interior—Bureau of Mines.

The supreme court based its decision on the case of one claimant, Pernovich, as the same questions were involved in all cases. Pernovich, before he was discharged, had worked steadily. He was not aware that he was suffering from any disease. The physical examination, however, showed that he had had silicosis for probably five years or more and was in the second stage of that disease and that tuberculosis had already developed. Although at the time of the examination Pernovich was able to perform his work, his chance of recovery would be diminished if he remained in the dusty employment in which he had been engaged. Re-examination three months later showed that his ability to do work requiring physical exertion was greatly diminished, if he could be said to be able to do it at all. Nevertheless Pernovich's employer and its insurance carrier contended that he had suffered no compensable injury in the course of his employment with his employer, the foundry company.

Disability within the meaning of the statute, said the supreme court, occurs when an employee is disabled from rendering further service; that is when he no longer has physical ability to perform his work in the usual and customary way. Unless he is so disabled he sustains no compensable injury even though in the course of his employment he is subjected to conditions that contribute to disability as an end result. The right of an employee to compensation depends on whether or not, in the course of his employment and because of it, an occupational disease renders him incapable of performing his customary work. It has been argued, said the court, that if an employee is entitled to compensation, as he is when he voluntarily ceases work because of physical incapacity to continue, then an employee who is discharged by his employer because of potential physical disability is equally entitled to compensation. For the latter contingency, however, the present law, which was framed to cover injuries resulting from accident, rather than incapacity resulting from occupational disease, affords no relief. An employee who has been exposed to industrial hazards, who has suffered no "physical disability" that prevents his performing his work in his usual and customary way, and who is discharged because an examination discloses that such disability may arise in the future, is not entitled to compensation for "medical disability."

It has been argued that it is against public policy to permit an employer to discharge an employee who has been exposed to industrial hazards, in order to anticipate his employee's future disability. That, however, said the supreme court, is a problem for the legislature and not for the courts. The courts can do nothing more than interpret and administer the statute. The remedy lies solely with the legislature. Finding no evidence to show that the claimants suffered disability within the meaning of the law during the period of their employment with the foundry company, nor until after the relation of employer and employee had been terminated, the judgments of the courts below in the several cases were reversed and remanded with instructions to enter judgments setting aside the awards of the industrial commission.

According to an insurance editor [3]"fear born of incomplete knowledge is the heart of the silicosis problem." He states that workmen's compensation insurance has been a headache to casualty-company executives for the past few years, although for the time, at least, the pressure has been lifted by the rise in industrial activity with an accompany increase in payrolls and premiums. Despite this, however, the future looks pretty dark to many, the reason given in two words is "occupational disease" and in one—"silicosis." In discussing the problem and the answer the editor states:

In considering the silicosis problem too often those affected have given way to semi-hysterical speculation on the possibility of disaster. Of course, the proper approach to an answer is to ascertain the known facts and from them consider the probabilities. The best program to bring about the least damaging of the probable developments can then be worked out, and an intelligent effort made to avert serious trouble. Let us first consider the picture as painted in its darkest tones by a partly informed and very worried insurance executive. He has been advised that 25% of all the industrial workers in the United States have silicosis in some degree. He has been told that silicosis is progressive, that there is no cure, and that death or permanent disability is inevitable. He sees, in many States, determined efforts to put silicosis under workmen's compensation, efforts that were successful, at the last session of the legislatures in New York and North Carolina. From past experience with the industrial commissions and other agencies administering the various compensation laws he has no confidence in their ability to distinguish between a workman disabled because of silicosis and a workman who, after exposure to silica dust, has x-ray indications of that exposure but no disability. Appalled by the possible accumulated liability, with the firm conviction that an effort to avoid all risks with a dust hazard will result inevitably in State compensation funds, he stands bewildered, waiting for the fast approaching storm to sweep away the compensation business. The final outcome may be as bad as, or worse than, is feared. If it is, the blame will be placed squarely on the shoulders of the industrialists and insurance men. Consider briefly the facts known about silicosis, as set forth by the best-known authorities. It is a disease of the lungs caused by the inhalation of large quantities of silica dust over long periods of time. In its early stages it is not disabling, and even in advanced cases disability or death is almost invariably due to some other infection of the lung, usually tuberculosis. A person with silicosis has less resistance to a tubercular infection than another with normal lungs, and an inactive tubercular infection may become reactivated by exposure to silica dust. Industry must remove tuberculars from occupations exposed to dust and will probably have to pay them compensation. The incidence of tuberculosis among those exposed to dust, outside of miners where other factors are largely responsible, is not alarmingly high. Modern engineering can reduce the dust hazard in any industry to the point where the occupation is perfectly safe for any worker whose lungs are free from infection when he begins work. Persons working under dusty conditions for years, who have developed some silicosis but not any infection, will probably never become disabled from this cause if the necessary protective measures are taken where they work. There is no medical evidence that silicosis causes the impairment of any other organ, such as the heart. The reserve lung capacity is so great that the lung can continue to function even though great masses of fibrous tissue have been formed as a result of silicosis.

With this in mind, take the hypothetical case of a foundry with 600 employees. Of these, 300 have been exposed to concentrations of dust varying from light in the case of furnace tenders to very heavy in the sandblast room. On medical and x-ray examinations it is found that almost 200 have some evidence of silicosis, with perhaps a dozen advanced cases uncomplicated by infection. Six have tuberculosis as well as silicosis. The risk is obviously dynamite, but the size of the explosion depends on how the situation is handled. The dust hazard in the plant must be cleaned up; to do this the cooperation of the employer is necessary. Then the employer will be expected to explain the exact state of affairs to the tubercular employees and send them to a sanitorium, with compensation paid to their families or dependents. Further, the men with advanced cases of silicosis will expect to be given light work within their capacity and kept on the payroll. The others can be left undisturbed, except that they should be required to take yearly physical examination; and all new employees will first undergo a complete examination. Such a program costs money, but if followed it will result in not only cleaning up a bad situation, but also in guarding against future trouble, and it is believed will in the end be less costly to the employer. On the other hand, if, after the results of the medical examination are known, the carrier gets off of the risk and does nothing, and the tubercular men are discharged, as well as those with advanced silicosis and some of the others, it is more than likely that the employer will be faced with suits which will turn the liability back on him and his carrier. At the same time the employer will find it impossible to get insurance from another company and he will join in the chorus of those shouting for a State fund. The big problem in silicosis is education. Manufacturers must be convinced that the hazard exists and that it must be eliminated. The medical profession, now woefully at sea, must learn how to diagnose silicosis and to determine the extent of disability, if any exists. Industrial boards and commissions must study the disease, its

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causes and effects, so that they will be in a position to make an intelligent decision on the medical evidence. The insurance companies must learn how to attack a risk which has a silicosis hazard, so that it may be made insurable, and how to handle it when it is insured. The refusal to write any risks with a dust hazard and the cancellation of any now insured will not solve the problem and will probably destroy the compensation business.

Sherman[1] has pointed out the lack of experience with compensation for silicosis or other dust diseases of the lungs under schedules in the United States—which foreign experience indicates calls for special terms and conditions to safeguard against impositions. He states that in Connecticut, Massachusetts, California, and Wisconsin the law nominally has long covered silicosis but in each of these States has had a peculiar history, significant enough to be noted in some detail.

In Connecticut[4] the original compensation law covered only injuries that happened at a definite time and place, that is, by accident. In 1919 the law was amended to abrogate that limitation and was then construed by the courts to cover all physical ill effects of working, whether resulting suddenly or gradually and whether the usual, expected, healthy or unhealthy accidents or conditions of employment, including the results of aggravations of ill health due to predisposition, pre-existing diseases, vices, and bad habits or bad living conditions were immediate or contributing causes. The result was to stretch the compensation law far into the fields of ordinary health, invalidity, and life insurance, to induce an intolerable flood of claims, and to lead to a radical reaction. In 1927 the law was amended to cover only "accidental injuries" and "occupational diseases", to exclude all injuries unless due to employment "otherwise than through weekened resistance or lowered vitality", and to reduce the compensation benefits in cases of aggravation of pre-existing diseases. The Connecticut law as amended has been operating quite smoothly to cover a wide range of diseases generally recognized to be "occupational", including silicosis; but the limits to its coverage and the conditions to the right to compensation under it have not yet been exactly defined and are still subject for litigation and uncertainty.

In Massachusetts[4] the compensation law enacted in 1912 nominally covered all "personal injuries" arising from and in the course of employment; but the meaning and content of the term "personal injuries" was a subject of uncertainty and continual litigation until it was decided in 1929 that disablement from inhalation of silica dust is a compensable injury. Claims for compensation for silicosis, however, were rare until "claim consciousness" was aroused in 1933 by publicity given to the dismissal of 42 employees from a foundry at Taunton because upon medical examination they manifested incipient symptoms of silicosis or tuberculosis. Since then conditions in Massachusetts have been chaotic, to the distress of all parties concerned. Many employers are having difficulty in obtaining insurance because unable or unwilling to pay the price demanded by insurance carriers for silicosis coverage, whereas the insurance carriers are reluctant to provide such coverage at manual rates for fear they will prove inadequate. In the Defilippo case[5] the court held that "an insurer under the workmen's compensation act must take an employee as he may happen to be during the term of the policy."

Since 1918 the workmen's compensation law in California has covered "any injury", including a disease arising from and in the course of employment. During the earlier years under this law silicosis was popularly unknown, and by the time it began to be recognized old cases were barred by the requirement that a claim for compensation for an occupational disease must be made within 6 months after the first manifestations of symptoms of the disease. In 1930 the construction was liberalized to permit a claim within six months after disablement. and in 1933 the supreme court still further liberalized the construction by ruling that in case of a progressive disease, such as silicosis, a claim is timely if made within six months from the date when the employee first learned or reasonably should have known that

his disability was due to such disease. That decision, according to Sherman[4], with its sweeping implications has stirred up a lively and widespread appreciation of the possibilities of gain through claims for disability from ill health attributed as an afterthought to exposures in employment. Consequently it may be stated that experience with compensation for silicosis in particular and other progressive "occupational diseases" in general is only beginning in California.

In Wisconsin the workmen's compensation law covered only "accidental injuries" or "injuries by accident" until 1919, when it was amended to cover "occupational diseases." Notwithstanding this amendment, claims for silicosis were almost negligible in Wisconsin until 1931, when unemployment became widespread and numerous claims were instituted. With data available for losses under policies expiring in 1933, there is now about three years' experience to indicate the results of applying the Wisconsin law to compensation for silicosis; and such experience is alarming.

Two hundred damage suits[6], filed by former workmen against the contracting company that built the Hawk's Nest Tunnel in West Virginia on the ground that they had contracted silicosis while employed in the construction of the tunnel, were eliminated by the decision in two of the cases that a century-old statute of limitations of the Virginia code of 1819 required that the action be brought "one year after the right to bring the same shall have accrued and not after." (The action was instituted in October, 1933; the time of employment was from April, 1930, to September, 1931.) Counsel for plaintiff asked for changes in the construction under "the rule of reason, the dictates of humanity, the principles of common justice, and the development and progress of the law in step with changing conditions." To this plea the court replied:

For more than a century legislatures, presumed to know the judicial construction of the statute, have ratified the construction by not amending the statute. It would be unwarranted for us at this late day to introduce by construction any innovation not authorized.

According to a bulletin of the West Virginia Coal Association[7] rates charged subscribers in West Virginia for coverage in the workmen's compensation silicosis fund will be measured by the degree of exposure to the dust hazard with an allowance for dustproof equipment.

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The Reduction of Absenteeism

A CERTAIN amount of absenteeism is inescapable and sickness should be the major cause of it, so that is the aspect to which I shall confine myself. I do not want to consider it from the purely medical aspect, but to go over some of the main points which, as an industrial doctor, I have found will affect sick absenteeism.*

Unfortunately there is no exact standard known by which we can measure whether the amount of sick absenteeism in any particular works is lower or higher than can reasonably be expected. The National Health Insurance authorities state that in 1934, 28 million weeks' work were lost through sickness, or an average of 14 days per annum per insured person. It must be remembered, however, that these figures include cases of very long sickness in persons who by reason of their infirmity are probably unemployable. They also include the whole of Saturdays and Sundays, which are not usually included in industrial records, so that for these two reasons are probably higher than would be reasonable to expect from any firm. On the other hand, they do not include any sickness of under three days' duration.

The Industrial Health Research Board has been working for some years on sickness figures, but has not yet published its results. They stated, however, in their report of 1932 that they had found 5.6 working days per head per annum to be the average of the records they had examined.

In spite of this uncertainty as to the exact figure a good sick rate should be, I think it is worth while to try to keep a record of time lost due to sickness and from time to time to study it carefully. The form in which it is kept will naturally vary in different firms, but I think the best way for comparison purposes is to express the result in working days lost per head per annum, as both the National Health Insurance and the Industrial Health Reasearch Board give their results in this way.

In any method of keeping sick records there are three very obvious sources of error. First of all is the length of time a long case is kept on the books. The practice of removing a sick person's name from the books after two or three months' absence is quite wrong if a true record is wanted. Secondly, the amount of short time worked, if any: if a works is on one week in three short time and a worker goes sick for three weeks, is that to be counted as two or three weeks' sickness? It is certainly only two weeks' lost time, but it is three weeks' sickness and should be recorded as such in a sickness record. The third snag is the fact that with a small group of workers under consideration one or two long cases of illness will upset the rate altogether and put it up so much that an entirely fictitious result is obtained.

When a reasonably accurate sick rate has been obtained, the thing we all want to do is to find out whether or not it can be reduced. Obviously it can be reduced if every sick employee is discharged, but needless to say, this is not a method any self-respecting firm would adopt. The amount of time which an employee can loose without running the risk of dismissal varies enormously in different firms, but if we are faced with a labour shortage we must be prepared to give sick employees a long rope. From a social point of view one need hardly say that dismissal on health grounds should be avoided whether there is a labour shortage or not.

If a sick rate is under three days per head per annum I should be inclined to view it with grave suspicion. I would investigate very carefully the method of record keeping, the labour turnover, and the efficiency of this apparently exceptionally healthy group of workers.

CAUSES OF A HIGH RATE: Causes entirely under the control of industry: The more usual problem is how to reduce a sick rate which seems higher than one might reasonably expect. There are certain factors which will affect a sick rate which are not entirely under the control of industry, and these should be considered first. Perhaps most important of these is the economic status of the workers and their housing conditions. It is not reasonable to expect the same standard of health from the inhabitant of an overcrowded tenement house who is striving to keep a family of six or more on an insufficient wage as from the inhabitant of a model house on a healthy housing estate who is keeping a family of three or four on a higher wage. The poorer worker will be forced through sheer necessity to work on through many a minor illness which would quite rightly incapacitate the better off worker, but his efficiency and his health will inevitably suffer sooner or later. I have seen this happen over and over again in our own factory girls. A girl works without losing a day for years; then she gets an attack of flu and she just cannot recover; or a girl who has been a good worker for years gradually gets slow or careless. Then one finds that she has perhaps been supporting a widowed mother and one or two younger brothers and sisters for years and lives in a slum. She has been using up her small stores of reserve strength during the whole of her working life, and has none left to cope with the added strain of an illness which would have incapacitated a more fortunately situated worker for a fortnight.

The age and sex of workers will affect a sick rate. Women generally lose more time than men, but I doubt very much whether it is because they have more ill health. If the disproportion between men and women is more than about a day a head per year I think the supervision the women are under should be scrutinised, as girls seem to react much more quickly to unwise supervision than do men. Of course, the fact that very often girls have no dependants will made it more possible for them to stay off for a com-

* DR. M. HOSSELL, Medical Officer, Rowntree & Co., Ltd.; paper delivered at a Conference organized by the Institute of Labour Management at Harrogate, April 25, 1936; published in *Labour Management*, June, 1936

paratively mild illness than it would be for a man with a family to keep.

Young workers generally lose days and half-days more than adults, but as a rule have fewer long illnesses. Elderly workers on the whole do not lose many half-days, but if they go off sick at all are usually slow in recovering. I do not believe in pampering young workers, but it should never be forgotten that the first year in industry is inevitably a great physical and nervous strain on an adolescent, and anything which can be done to temper the wind to the shorn lamb will be rewarded by better health, greater efficiency and less absenteeism. The child of 14 who comes straight to industry from school has to adapt itself to a change in working hours from about 30 hours a week to 44 or more. He has to do work which is often very dull and uncongenial and begin to assume the responsibilities of adult life, and he has to do all this during a period of growth both physical and mental. It is small wonder that this growth, which is a gradual process, cannot always keep pace with the added strain which has been so suddenly imposed. Adaption to industrial life has got to take place if the adolescent is to hold his own in life, but the more gradual this adaptation can be made the more useful men and women we shall eventually turn out.

The type of work to be done may affect sick absenteeism and it is not always in the power of the industry to modify it. Other things being equal, a light sedentary worker should lose less time through sickness than one on heavy or outdoor work. An indisposition, such as a sprained left wrist, would be bearable for a clerk while it would totally disable a bricklayer. It must be remembered though that a weakling may have learnt by experience the futility of attempting heavy work and so seek lighter employment, and so sedentary workers are often less robust than heavy workers. If, however, your light workers do not consist of people with known physical disabilities and they lose more time than heavy workers of the same sex, there may be something wrong with their conditions of work.

East of transport to the works will affect a sick rate chiefly in respect to casual sickness. One expects more lost time from workers living far from the works with inadequate methods of transport than from those living near at hand. A worker with a cold may feel he can manage to get in a bus which will take him from his house to the factory and then do a day's work, but he will think twice about it if he has to start out on a rainy morning for a six miles' cycle ride with streaming eyes and a running nose.

Short time will diminish a sick rate without necessarily improving the health of the workers. Some short illnesses will be convenient enough to occur during a short time week; also if a worker has something like a bad cold two or three days before he is due to go on short time he will stick at work buoyed up by the knowledge that he is bound to have a rest soon, when had he been on full time he would have gone off. Very heavy short time may increase a sick rate without necessarily any deterioration in health because sick pay may be more than short time pay, and there is no financial inducement to recover quickly from an illness.

2. Causes under the control of industry: The next factors we should consider when seeking for the cause of a high sick rate are more directly under the control of the industry. There are two ways by which a sick rate can be reduced. The incidence of sickness can be reduced and the recovery time can be accelerated: in other words we want fewer people to go off for shorter times.

The first factor to be considered in reduction of the incidence of sickness is the hygenic condition of our works. We all know perfectly well that no one can work and keep well and efficient without a sufficiency of light and air. Temperature matters much less. The human body can fortunately adapt itself to wide variations in temperature without harmful effects, but no one, however robust, can keep good tempered, alert, efficient and healthy in a badly ventilated room. The Kata thermometer is simple to use and is the best method I know of testing the ventilation of a room by ascertaining the cooling power of the air. The Medical Research Council has published an excellent account of this instrument from which all details can easily be understood. The generally agreed standard is that no reading above 6 should be regarded as satisfactory if the outside temperature is 70° or less and no reading above 5 if the outside temperature is over 70°. I do not want to say more about hygienic conditions, because we all know so well what we should aim at, but I would like to stress the importance of putting first things first. It is no use hoping to have healthy workers if they have to work in dark, badly ventilated rooms, and the most costly and elaborate welfare schemes will not improve health if working conditions are bad.

The next factor to be considered in reducing the incidence of sickness is the presence of any known industrial hazard. We have first of all to satisfy ourselves that this hazard is really necessary at all, and if it is, whether we are doing all that is in our power to minimise it. The sickness records of all employees who are on work where there is a known industrial hazard should be very carefully watched. Do not just look out for the particular disease which might arise, for most of these diseases are more likely to occur in a debilitated than in a healthy person. This is true of occupational dermatitis, which I have found several times starting after a quite irrelevant illness in a worker who has previously been on the same work for years and never had dermatitis before.

That long hours of work increase the incidence of sickness few people would dispute, and yet it is astonishing the way overtime is often worked without taking this well-known fact into consideration. If heavy persistent overtime is worked, absenteeism will inevitably go up sooner or later, and then because there are fewer people to do the work, more overtime is added and so it goes on in a vicious circle. I quite realize the apparent inescapability of overtime in certain industries at times, but we should never allow ourselves to forget that if prolonged it is invariably worked at the cost of the health and efficiency of the workers.

PREVENTION OF ACCIDENTS: Prevention of accidents will decrease the incidence of sickness. Prevention of machine accidents is partly a matter of good guarding and so is the engineer's job, but the human factor plays a very large part. I have vivid recollections of saying one afternoon at 2:45 that a certain machine was foolproof and treating a girl at 4 p.m. the same day who had managed to get three fingers caught in this same machine. Some people certainly seem to be accident prone, and when such people are known they should not be allowed to come in contact with machinery. The difficult thing is to detect them before they have damaged themselves or their fellows. There are certain things to be looked for in a medical examination and many psychological tests for accident proneness, but there are some things which any layman can detect. First of all, look for any physical defects, especially of the hands, such as a stiff finger. Then watch the person walk to see whether there is a limp or any peculiarity of gait. These points seem elementary, but I am reminding you of them because judging from some of the applicants for employment I have seen who tell me they have been on machine work for years and generally have a long history of accidents, I can only think that they are forgotten. Then look at the eyes: a person with a squint is unlikely to have good binocular vision. It is very easy to do a rough test of vision with a Snellen test card, and I think it is a think an employment officer might very profitably do when engaging workers for complicated machines, and as it only takes about a minute per person, it would not lengthen the time spent on interviewing very much.

Facilities for getting a decent meal should help to diminish the incidence of sickness. Many workers will not get up early enough to have breakfast before starting out, and if there is no chance of getting a good meal during the day their fatiguability will be increased, and with it their tendency to illness.

The last factor which I would consider in reviewing the incidence of sickness is the amount of casual absenteeism and the firm's attitude to it. There are very few complaints which really incapacitate a person for a half or one day. I can only think of four—a bilious attack, shock after an accident, migraine, and women's ailments. Generally speaking, if a person is off for a day with anything else, either he should have stayed off longer or he need not really have stayed off at all. A cold is rarely a justifiable cause of a day's absence, though it should much more often be a cause of three days' absence than it is. The average person who has a cold does not cure it with a day at home; he just feels a bit better and then returns to work

and infects his co-workers. I would like to send everyone who is starting a cold off work for three days, but if I find a person who has stayed off for a day or half a day I usually think he might just as well have stayed at work for all the good it will have done him, or in diminishing the risk of infection. There is one reason for a day's absence which I think is perfectly justifiable, though it is seldom admitted by the worker, and that is frank tiredness. This is especially true of young and elderly workers. The Industrial Health Research Board state that "with the exception of the common cold there is no evidence that absence for some of the more minor ailments reduces the liability to the more serious ailments." While I quite agree with that statement, I do think that a very occasional day off for a young or elderly worker will often increase his efficiency.

A high sick rate is often due to a slow recovery rate rather than a high incidence rate. The time taken to recover from a similar complaint by different people is very variable indeed, and it is impossible to give a fixed time for any particular complaint which can be regarded as normal. It is not always easy to tell from the medical certificate what the absentee is really suffering from and even given a cut and dried cause like an appendicectomy the illness may vary in severity and the individual varies very much in his power of recovery. Recovery power is dependent on many things, many of them intangible and difficult to assess, but there are one or two ways in which it can be quite definitely helped by those in industry. The most easily demonstrable of these ways is by efficient first aid. A clean cut heals in a few days, a septic cut may take months. An injury caused by a dirty instrument need not go septic if it is properly treated from the very start, but an injury caused by an instrument which had just been taken out of a bowl of strong disinfectant will go septic if it is treated carelessly.

TREATMENT OF ACCIDENTS: We want to keep carefully in our minds what we are aiming at in our factory first aid or ambulance sections. If we simple intend to give first aid, we must do the minimum treatment compatible with safety and then send all cases on to hospital or to a panel doctor; if we mean to treat injuries more than once, we must be equipped to give at least as good treatment as would be given by any efficient hospital. If a factory has no medical service at all, it can best reduce its absenteeism from injuries by doing the minimum of first aid treatment on the premises and carefully insisting that every accident case is seen at hospital or by the panel doctor as soon as possible. No untrained ambulance worker should be allowed to re-dress a wound in any circumstances whatsoever unless he is under the supervision of a doctor or a nurse. The ambulance boxes prescribed by the Home Office for use in factories do not contain materials for continued treatment of any injury and were never intended for such use, and if that is all the equipment there is in a factory nothing but the strictest first aid should be attempted. If that is all that is done, such a factory must expect to get a fair amount of absenteeism from accidents which have happened both in the works and at home, but, if there is no meddlesome first aid and unskillful re-dressing done, the time lost by each case should not be unduly long.

It should reduce absenteeism from accidents markedly if there is some medical or nursing service on the factory premises, so that workers can have injuries attended to and remain at work, and the more complete their service the greater should be the reduction in absenteeism from injury.

Recovery rate will be accelerated by giving workers some facilities for light work or shorter hours on return from more serious illnesses. I have time and time again persuaded a worker to return to work after a long illness by suggesting shorter hours for a time, and I am sure that on their return they get completely well much sooner than they would have done if they had remained off work. It takes a lot of courage to face an eight or nine hour working day on heavy work after a month or more of illness, and leniency and consideration to workers who have been seriously ill can better reduce incapacity if wisely used than almost anything else. There is a large psychological factor in very many cases of long absenteeism whatever the initial cause of the absence, and it is this factor, often of fear, that we can modify by making return to work easier. Generally, I have found it better whenever possible to bring a sick person back to shorter hours on his old job than to lighter work for full days, but of course in many case the latter way is the only feasible one.

When we have carefully gone over all the factors which might influence our sick rates we must then realize our limitations and remember that there are at least as many if not more factors outside than inside the works which may affect the health of our workers. A factory should never be a forced labour camp, nor should it be a psychological clinic, and if we honestly do our best to cope with all the reasons for sickness which are related to the works we shall have little time left to meddle in extraneous causes. I think we should do more harm than good if we attempted to investigate or interfere with the lives of our workers except in those cases where we could definitely help, remove, or improve the factors which may give rise to ill-health or unhappiness.

Control of Lead Poisoning

MICHIGAN reports great progress during the past year in the control of lead poisoning hazards in the automobile industry, perhaps the most important industrial hygiene problem in the State.* The State Department of Labor and Industry, working in cooperation with leading manufacturers, has done constructive research in the field and achieved the installation in many plants of effective protective devices.

An outbreak of lead poisoning occuring in the automobile industry during 1934 and 1935 is said to have been the most serious epidemic of the disease in this country during the past decade. Relatively free from lead hazards since dry-sanding of lead-painted wooden bodies was discontinued (the last epidemic from that cause was reported in 1934), the industry in recent years has found itself faced with an even more serious occupational disease problem arising from the filling in, with a lead-tin alloy, of welding depressions and other indentations on one-piece, all-metal bodies. The difficulty lies in the control of the lead dust and fumes released by the use of lead pots and torches, and by processes for finishing the leaded metal surfaces by power grinding, hand filling, sanding, etc.

As a result of this exposure there has been a high incidence of lead poisoning, while in the case of many workers who suffered no apparent injury and lost no time from employment, the fact of lead absorption has been established. According to one estimate as many as 4000 workmen may have been injured to some extent during the 1934-35 production season.

Dr. Carey P. McCord and associates, in a report published in the American Journal of Public Health in October, 1935, stated that during the past 10 years there have been many affirmations that industrial lead poisoning as a waning disease. While in some measure this has been true up until 1934, the outstanding fact is that it is the severity of the affection that is waning, rather than the frequency. Very strikingly this was borne out by the 1934-1935 epidemic. As far as is known among the large number of cases at that time, there were no proved deaths, little profound encephalitis, very few instances of wrist drop or foot drop, etc. The report continues, "with certainty it may be believed that there have been milder degrees of neuromuscular injuries, encephalitis, etc. The outstanding and most uniform manifestations have centered about involvement of the gastrointestinal tract, accompanied by excessive fatigability and blood changes."

The activity of the Michigan Department of Labor and Industry, under Frank B. Wade, Commissioner, in checking lead hazards in the automobile industry dates from May, 1935. At that time all registered physicians in the State were requested to report immediately all cases of lead poisoning, and a number of experts in industrial medicine were retained to carry on an intensive research program. The initial survey of the situation is reported to have evidenced widespread consciousness on the part of manufacturers of the seriousness of the hazards and a real effort to eliminate them. At first respirators were relied upon to a large extent for employee protection; these proved not entirely adequate even when worn, and workers habitually discarded them because of discomfort unless strictly supervised. It finally came to be recognized that the surest way to control the lead poisoning hazard is to reduce the lead content in the surrounding air rather than depend on the efficiency of respirators.

Cases of lead poisoning were found

*Survey of Labor Law Administration, U. S. Dept. of Labor, Division of Labor Statistics, August, 1936.

particularly incidental to the use of disc grinders on soldered surfaces, and the department has disapproved this process unless the grinder is a wet grinder or vacuumized. The use of a vacuum hose instead of a sweeping broom is recommended by the department and is now in use in a number of plants.

In most plants today the air is tested for lead content every 30 days. The necessity of keeping the lead content in the breathing atmosphere below a recognized permissible concentration is now widely recognized throughout the industry.

Ventilating systems have been installed to achieve this condition of the air; the cost in some instances in the larger plants has exceeded $50,000.

Nearly all plants employ a 30-day medical check-up of employees to detect evidence of absorption before definite poisoning occurs. Lead absorption is revealed by blood examination, and where the test shows over 1.5% basophilic aggregation, the employee is removed from the lead area, and the condition causing the same is corrected.

The necessity for personal hygiene in this branch of the industry is being given more attention than ever before. The importance of this phase of the lead problem cannot be overemphasized. Strict supervision, to enforce rules pertaining to hygiene, has been one of the consistent recommendations of the Michigan department. The custom of hanging street clothes, storing and eating lunches in lead areas is now generally prohibited. The problem of educating employees to change lead dust-laden work clothes at least weekly, and to recognize the importance of refraining from chewing tobacco and similar habits during working hours is only indicative of the wide scope of the problem and the necessity of full cooperation of employer and employee to eliminate the menace.

The Department of Labor and Industry has also given its attention to problems related to and arising out of the existence of the lead poisoning hazards. Of these, perhaps the most important is the rehabilitation of the affected employees. Most of the manufacturers pay a weekly compensation of $18 per week while the workers are disabled. On their return, employees are assigned to light work at their regular wage rates until physically fit to resume metal finishing. A few exceptions to the above have been taken up by the Department and the above results were always obtained.

A system of basophilic aggregate test for all metal finishers is used by practically all employment offices of the plants when hiring metal finishers. This brings up the problem of employees unable to get work because of lead content. The Michigan Department of Labor and Industry feels this may develop into a real problem especially if lead cases are recurrent. They consistently recommend that all employees be given a test for lead before being laid off, which is being done in a large number of plants. It can be said that an intensive, intelligent, cooperative effort has been put forth by the Michigan Department of Labor and Industry to control this evil, which possibly may only be eradicated by means of finding a substitute for solder in the manufacture of all steel automobile bodies. Care should be taken in any decision regarding a substitute and before another substance is acacepted in place of lead, detailed knowledge regarding the health hazards associated with its industrial use should be available.

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